THE BRITISH JOURNAL

OF

OPHTHALMOLOGY

APRIL, 1936

COMMUNICATIONS

"ATYPICAL, INDIRECT, INCOMPLETE RUPTURE OF THE SCLERA"*

BY

A. TILLEMA, M.B.(Edinburgh and Leiden), M.D.(Amsterdam)

IT will be remembered that indirect ruptures of the sclera occur most frequently on the inner and upper aspect of the globe (Müller, Wagenmann) and that ruptures in this region are termed typical. This typical localisation is interesting, but as theories explaining it are fully discussed elsewhere (*e.g.*, Wagenmann, Lister) they need not be repeated here. It will also be remembered that these ruptures originate in the neighbourhood of Schlemm's canal and that they continue backwards and outwards along Schlemm's venous plexus to reach the outer surface of the sclera some millimetres behind the corneo-scleral junction (E.Fuchs). In young subjects, on the other hand, the rupture may not pass backwards but directly outwards into the corneal limbus or even forwards into the cornea (E. Fuchs, 1905). This last type of rupture is usually small. Both varieties run parallel to the corneal limbus, a course influenced by Schlemm's canal and also perhaps by the direction of the scleral lamellae, which have been shown to split in a circular direction between the limbus and the insertions of the recti muscles (Kokott, 1935).

Although these typical and complete ruptures are fairly frequent one does not find many descriptions of either incomplete or

* From the Department of Ophthalmology of the University of Amsterdam (Professor W. P. C. Zeeman, M.D.).

atypical ruptures. A combination of both, *i.e.*, atypical, incomplete ruptures, has not yet been described in the literature.

FIRST CASE.—A.J.K., aged 17, stumbled and knocked his left eye against the corner of a wooden box. Six weeks later the globe was removed to relieve the patient of painful traumatic glaucoma.

Microscopical description. Superiorly the scleral fibres superficial to the pectinate ligament are pale and undulating. The area where this is observed runs backwards and outwards from Schlemm's canal along the venous plexus. Some of the scleral lamellae are interrupted and the interlamellar cell nuclei are pale and swollen. (*Partial, typical rupture of the sclera,* Fig. 1).

Also superiorly, but farther back, analogous changes are seen. These occupy an area between the pars plicata of the ciliary body on the one hand, and the insertions of the recti muscles on the other. The scleral fibres in this region are even more wavy than outside the pectinate ligament, they are paler and seem to have been broken in more places. The affected area begins in the inner layers of the sclera on the lateral aspect of the globe. Thence it passes medially and more superficially, and reaches the outer sclera in front of and behind the insertion of the medial rectus muscle. In this region the pathological changes occupy the whole thickness of the sclera and here the sclera is thinnest. In general the inner layers of the sclera have been principally affected. (*Atypical, partial rupture of the sclera,* Fig. 2).

The innermost layers of the sclera contain a few cells with long, slender nuclei here and there.

Inferiorly, just outside the point of entry of a vortex vein, the scleral fibres show similar changes. In addition, small round cells lie scattered between them. These changes are situated first on the outside, then on the inside of the vein, and are observed only in the inner third of the sclera. The same is seen around the ciliary vessels.

SECOND CASE.—Th. de L., aged 30 years, received a blow with a piece of wood on the left eye. The globe was removed eight weeks after the accident to relieve the patient of painful traumatic glaucoma.

Microscopical description.—The tissue spaces of the episclera are filled here and there with yellow pigment granules, especially around one of the vortex veins. Thence they can be traced into the spaces surrounding the vein in the sclera. They can also be traced backwards towards the posterior pole of the globe. These granules contain iron.

The sclera is thin in its anterior part and appears to have been stretched. Superiorly, but close to the equator, the outer scleral fibres are a little wavy and pale. These pathological changes are

FIG. 1.

Partial, typical rupture of the sclera running backwards and outwards from Schlemm's canal.

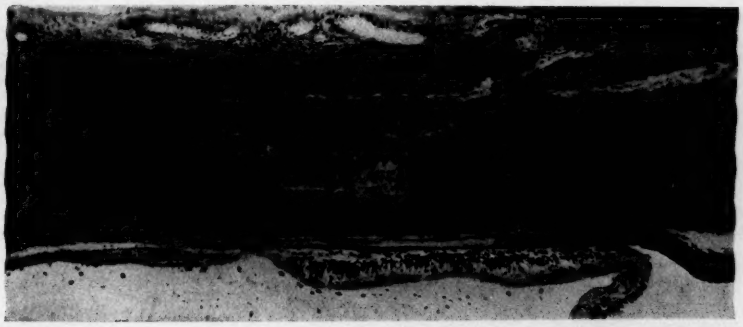


FIG. 2.

Partial, atypical rupture of the sclera. Above, the normal, darkly stained tendon of the rectus muscle is seen, contrasting with the abnormal paleness of the sclera.

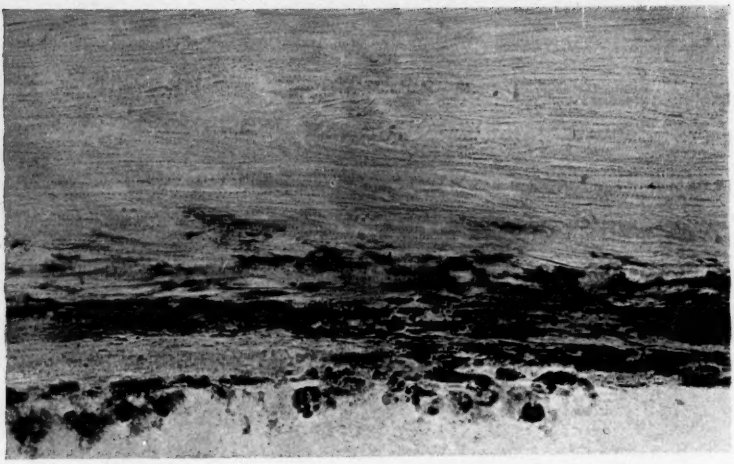


FIG. 3.

Rupture of innermost layers of the sclera indicated by the presence of iron (the dark "smudges" outside the choroidal pigment).

FIG. 4.

Atypical, partial rupture of the sclera. Note the obvious scar-tissue formation in the sclera and the destruction of choroid and retina.

seen medially and laterally as far as the insertions of the recti muscles, but are most marked medially and above. A fairly large number of spindle cells with well-stained nuclei and a small number of round cells are interspersed between the scleral fibres. No iron can be demonstrated in this area. In its innermost layers, however, the sclera is found to contain a surprising amount of iron with special staining (Fig. 3) although with ordinary stains only a few spindle cells and a few pigment granules can be demonstrated.

Also superiorly, but more forward, the sclera is thin, and irregular in structure throughout its thickness. A large amount of haematogenous pigment is scattered through all this area. Superiorly and medially the inner layers of the sclera are markedly pale and contain a striking number of spindle cells with well stained nuclei. In this latter region retina and choroid have been completely destroyed (Fig. 4).

Summarizing, one may say that in this case pathological changes are found in both outer and inner layers of the sclera. In the outer layers no haematogenous pigment is found, and for this reason it seems unlikely that the findings in these layers are related to the accident. In the inner layers, and especially above and medially, haematogenous pigment is found in abundance as well as marked scar-tissue formation. Furthermore, the changes of the scleral fibres in this latter region are identical with those found in the first case. The second group of changes, therefore, must be related to the accident.

Discussion

In these cases pathological changes were observed in the inner layers of the sclera. There are two possible ways in which they may have arisen, (*a.*) through the raised intra-ocular pressure causing a staphyloma of the sclera, and (*b.*) through a partial rupture of the sclera. When a staphyloma is being formed however, the sclera contains many cells and many young vessels, (Elschnig 1928); this is a reason for thinking ruptures of the scleral fibres more likely. But there are other reasons even more convincing. The changes are found on the upper, inner aspect of the globe, a localisation typical for indirect ruptures of the sclera. Also, the changes occur mainly in the inner layers of the sclera and this again is typical for incomplete ruptures. Finally, the presence of haematogenous pigment in the inner layers of the sclera can only be explained by previous haemorrhage. Therefore we may safely assume that the microscopical findings in these two cases are based on partial rupture of the sclera. Two different types of rupture were found, *viz.*, a typical rupture near the corneal limbus

in the first case, and an equatorial (*i.e.*, atypical) rupture in both cases. The latter type of rupture appears to be very rare indeed.

Atypical ruptures have not often been noticed. The equatorial are very rarely encountered in civil practice. Wagenmann (1915) mentions cases by Wadsworth, Montagnon, Ask, Nuel, Purtscher, Weeks, Oeller and Axenfeld. More recently cases have been described by B. Schaefer (1926) and Casanovas (1933). According to von Szily (1918) and Lister (1924) they are much more frequently found in military practice.

Typical, incomplete ruptures have also been infrequently described. Although a certain number of them are found in the literature it must be admitted that in many cases the diagnosis is open to suspicion. It must not be forgotten that for a sure diagnosis a microscopical examination is indispensable (Müller; Wagenmann; E. Fuchs, 1926). If, therefore, one insists on this for a correct diagnosis all the following cases that are based on clinical examination must be disregarded: Von Arlt (near the corneal limbus; quoted by Müller and by E.Fuchs), Müller (near limbus), Bertram (near limbus), E. Fuchs (1905, near limbus), Purtscher (quoted by Wagenmann), E. Fuchs (1911, one case near limbus and another case 7 mm. behind the limbus).

Even some of the cases that have been examined microscopically cannot be accepted with certainty. H. Schäfer has omitted to state whether the whole rupture has been examined in his case. Inasmuch as a considerable hypertrophy of episcleral tissue is mentioned in the description, one is led to think of connective tissue formation at the end of a complete rupture of which the author may have examined only one end. Buchanan's description is too brief to be convincing.

One may state therefore that only von Szily's, Fuchs's and Burk's cases are described in sufficient detail to warrant a sure diagnosis. All three cases constitute typical, incomplete ruptures of the sclera. Burk's case differs from the other two, however, as it belongs to the group of small ruptures described by E. Fuchs in 1905. To this small number of incomplete, typical ruptures the anterior rupture in the first case of my article may be added, bringing the total up to four. Inasmuch as atypical, incomplete ruptures have not yet been recorded, the two instances of incomplete, equatorial rupture are new. In both cases it was mainly the inner layers of the sclera that were affected. This agrees with what is known of ruptures in general, although Müller in his monograph expressed the (often misquoted) opinion that the sclera may tear from without inwards just as well as the reverse. However, Müller's theory has been disproved by the findings in cases of incomplete rupture and in cases where the rupture, although complete, was very small (*e.g.*, Wintersteiner).

In every case the inner parts of the sclera were more extensively torn than the outer.

The only description of an incomplete equatorial rupture that I could find in the literature was published by Axenfeld (1899). Probably, however, this must be regarded as a direct rupture, as the patient, a lunatic, had torn out his own eye. The globe was not examined microscopically, but macroscopically the inner layers at the equator were torn.

Although the literature yields no other cases of this type I believe that it is not so rare as this would suggest. Garnier (1891), for instance, gives a detailed description of traumatic glaucoma in a young boy whose eye had to be removed two weeks after the accident. Within this short period a staphyloma had developed superiorly and medially at the equator. Unfortunately the sclera was not described microscopically with the other parts of the eye, but if glaucoma had been the cause of distension of the sclera one would expect the sclera to distend generally, as in cases of buphthalmos. Inasmuch as in Garnier's case a staphyloma developed within two weeks after a blow, it is reasonable to suspect a traumatic lesion to the sclera. Moreover, the staphyloma developed superiorly and medially; and it will be remembered that in both my own cases pathological changes were most marked in the same region, and that the majority of scleral ruptures occur upwards and inwards.

The interesting changes in other parts of the two globes discussed above will be described in two separate articles, one on rupture of the lamina cribrosa[17] and a second on traumatic glaucoma.

BIBLIOGRAPHY

1. **Axenfeld.**—Ueber Luxation des Augapfels als Selbstverstümmelung bei Geisteskranken. *Zeitschr. f. Augenheilk.*, Vol. I, p. 128, 1899.
2. **Buchanan.**—A case of rupture of the pectinate ligament. *Trans. Ophthal. Soc. U.K.*, Vol. XXVII, p. 103, 1907.
3. **Burk.**—Beiträge zur Anatomie der erworbenen Linsenluxation und ihre Folgen. *Arch. f. Ophthal.*, Vol. LXXXIII, p. 131, 1912.
4. **Casanovas.**—Zur kenntniss der atypischen Sklerarupturen. *Zeitschr. f. Augenheilk.*, Vol. LXXX, p. 35, 1933.
5. **Elschnig.**—*Handb. der spez. pathol. Anat. und Histologie XI* (Henke-Lubarsch), 1928.
6. **Fuchs.**—Ueber kleine Rupturen an der Corneoskleralgrenze. *Wien. klin. Wochenschr.*, p. 985, 1905.
7. ——— Ueber innere Skleralruptur. *Arch. f. Ophthal.*, Vol. LXXIX, p. 53, 1911.
8. ——— Regarding some unusual varieties of rupture of the sclera. *Ophthalmology*, October, 1913.
9. ——— *Lehrb. d. Augenheilk.*, 15e Aufl. Deuticke, Leipzig und Wien, 1926.
10. **Garnier.**—A case of traumatic glaucoma. *Wratsch*, Vol.XII, p. 636, 1891.
11. **Kokott.**—Das Spaltlinienbild der Sklera. *Klin. Monatsbl. f. Augenheilk.*, Vol. XCII, pp. 177-185; *idem.*, Vol. XCIV, pp. 33-45.
12. **Lister.**—Some concussion changes met with in military practice. *Brit. Jl. of Ophthal.*, Vol. VIII, p. 305, 1924.

13. **Müller.**—Ueber Ruptur der Corneo-Skleralkapsel durch stumpfe Verletzung. Franz Deuticke, Leipzig und Wien, 1895.
14. **Schaefer.**—Pathol. Anat. Befund einer atypischen indirekten äquatorialen Skleralruptur durch Kuhhornstosz. *Arch. f. Ophthal.*, Vol. CXVII, pp. 693-701, 1926.
15. **von Szily.**—Atlas der Kriegsaugenheilkunde, 3e Lieferung. Ferd. Enke, Stuttgart, 1918.
16. **Tillema, A.**—Anatomie en kliniek van traumatisch glaucoom. *Diss.*, Amsterdam, 1935.
17. ——— Partial rupture of the lamina cribrosa from contusion of the globe. *Arch. of Ophthal.*, Vol. XV, 1936.
18. **Wagenmann.**—Die Verletzungen des Auges. *Handb. d. g. Augenheilk.* (Graefe-Saemisch), 3e Aufl., 1915.
19. **Wintersteiner.**—Beiträge zur pathol. Anat. d. traumatischen Aniridie etc. *Arch. f. Ophthal.*, Vol. XL, ii, pp. 30-43, 1894.

VASCULARISATION OF IRIS AND CORNEA IN DIABETES

BY

DR. R. D. LAWRENCE *and* A. HAROLD LEVY

VASCULAR eye changes are frequent in diabetes, especially in elderly, long-standing cases, and are usually confined to the posterior segment of the eye, especially the retina. Much less frequently are found oedema of the posterior layer of the iris with ectopia uveae and occasional vascularisation of the iris, a condition of affairs which makes the eye unusually prone to iridocyclitis and glaucoma.

Vascularisation of the iris and cornea, such as we record, must be extremely rare and is not mentioned in the previous literature.

Diabetic History and Condition

Diabetes was diagnosed in 1923 in a slightly obese man, aged 49 years, home on leave from India. He had had no previous illness of any importance and no family history of diabetes. The hyperglycaemia and glycosuria were easily controlled by diet and no diabetic complications or organic abnormalities were present. There is no doubt that diet was not strictly followed and when next seen in 1926, heavy glycosuria, some ketonuria and a blood-sugar of 0·425 per cent. were found. Insulin was advised, but taken very irregularly and the patient was not seen again for eight years (May 1934). Fair general health had been maintained during that time although without doubt the diabetes had seldom been properly controlled. In 1933 the patient nearly died in India from a huge carbuncle, and in February 1934 from lobar pneumonia and empyema, when acute cystitis also developed. At this

time he was given 50 units of insulin a day and sent to England as soon as he was fit to travel. Two weeks before arrival he noticed his first serious eye trouble, becoming quite suddenly nearly blind in his left eye.

Physical Condition: May-December 1934. On arrival the diabetes was quite uncontrolled as he had had no insulin for 2 days—this is typical of the patient's attitude. The urine contained 5 per cent. of sugar, moderate amounts of ketones and the blood-sugar was 0·420 per cent. Moderate albuminuria and slight chronic bacillus coli infection were present.

The diabetes was complicated by severe peripheral neuritis—numb and painful feet and absent knee and ankle jerks—but his general condition was fairly good. Heart and lungs were normal, no marked general arteriosclerosis, pulses in feet good, and blood pressure 142/83 mm. The liver was slightly enlarged and smooth, not tender, but with a toughish rounded edge.

The details of the eye condition are described separately.

The diabetes was easily controlled by 40 units of insulin a day, with a diet of 130 grams carbohydrate: glycosuria and ketosis were absent, and the blood-sugar varied in the day from 0·1 to 0·200 per cent., being mostly at the lower level.

The general health and neuritis quickly improved and the patient was soon able to walk normally. In spite of this, the eye condition steadily deteriorated.

The following blood tests in October 1934 showed no particular abnormalities.

	Whole blood
Blood-urea	49 mgm. per 100 c.c.
Cholesterol	219 mgm. per 100 c.c.
	Serum
Total Protein	7·0 per cent.
Albumen	4·9 per cent.
Globulin (by difference) ...	2·1 per cent.
A/G ratio	2·4 per cent.
Fat (Ruckert)	0·68 c.c. per cent.
Calcium	10·2 mgm. per 100 c.c.

Ocular Condition

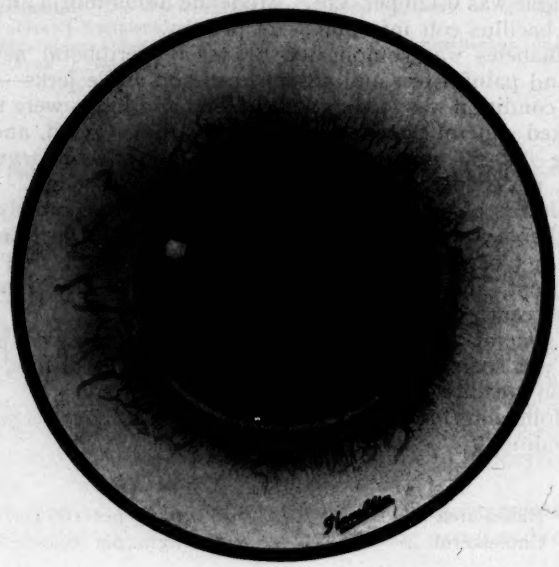
First seen about eyes on May 16, 1934, when the vision in the right eye was 6/9 partly and in the left counting fingers at half a metre.

Ophthalmoscopically: Right.—Veins much dilated, with local narrowings—few spots of yellowish exudate about the macular area and a few fine haemorrhages.

Left.—Gross haemorrhages all around disc and macula, but periphery fairly clear.

One week later the left eye became painful—the pupil was semi-dilated and the tension was raised. From the fundus there was only a dull red reflex and details could not be made out.

By the end of June, 1934, the tension in the left eye had increased. There was an hyphaema and fresh vessels were seen in



the posterior layers of the cornea and on the iris. The drawing shows the condition at that time.

By the early part of August, 1934, that is about seven weeks after their first appearance, the vessels on the cornea had disappeared. A small hyphaema remained—the iris was discoloured with many vessels on its surface. Dull red fundus reflex and bare perception of light.

By the end of August, 1934, this left eye became totally blind—with fixed oval pupil and many vessels on iris—fundus details could not be seen. The tension remained full, but not as high as it had been in June, there was very little pain now and when last seen in February, 1935, had remained the same.

The right eye remained more or less as it was when first seen until the end of August, 1934. The haemorrhages had become more numerous, but without much interference with vision.

On August 28, 1934, there came suddenly extensive haemorrhages in the central portion of the retina and many peripheral haemorrhages, and vision sank to counting fingers at 2 metres.

In this right eye the haemorrhages were confined to the posterior segment, but glaucoma became a dominating feature and on the suggestion of Mr. Whittington a paracentesis was performed on November 6, 1934. This relieved the pain for a time, but the tension gradually increased again and the eye became totally blind.

He was last seen on February 22, 1935, just before his departure for India and then he was having transitory attacks, at infrequent intervals, of pain in both eyes, but was completely blind.

The peculiar feature of this case is the development of vessels on both surfaces of the cornea and in a sense more remarkable still, their complete regression. No record of a similar happening has been found and it seemed worth while to record this.

TRACHOMA:

An investigation into the question of the presence of Bacterium granulosis (Noguchi) in Cases of Trachoma in Glasgow

BY

S. SPENCE MEIGHAN, B.Sc., M.B., Ch.B., F.R.F.P.S.G.

SURGEON, GLASGOW EYE INFIRMARY. OPHTHALMIC SURGEON, STOBHILL HOSPITAL, GLASGOW

AND

MARY URQUHART, M.B., Ch.B.

BACTERIOLOGIST, STOBHILL HOSPITAL, CORPORATION OF GLASGOW

HIDEYO NOGUCHI working at cases of trachoma among the American Indians at a reservation in New Mexico isolated a hitherto undescribed bacillus to which he gave the name *Bacterium granulosis*. Papers giving the result of this work were published in 1928, (The Etiology of Trachoma, Hideyo Noguchi, *Jl. Exper. Med.*, XLVIII, Supplement No. 2).

Since that time a mass of literature has appeared concerning the presence and aetiological significance of this micro-organism. A point which emerges is that different investigations show much diversity regarding the presence of the bacterium in cases of undoubted trachoma.

In view of this we set ourselves to answer the question whether or not *B. granulosis*, (Noguchi), was present in cases of trachoma occurring in Glasgow. The material investigated was obtained

from patients admitted to the trachoma wards under the charge of one of us (S.S.M.) at Stobhill Hospital. The patients were representative of those attending Hospitals and living in Institutions in and around Glasgow. All were early cases.

The general lines of the laboratory investigations followed those indicated by W. C. Finnoff and P. Thygeson, (*Arch. of Ophthal.*, V, 527, 1931).

In our work on the earlier cases, human blood agar was employed and was found quite satisfactory; as it was more convenient to obtain horses' blood, as advocated by Noguchi himself, this was used in the later cases and was proved to be equally satisfactory.

Further, the presence of cocaine inhibited the growth of *B. granulosis* and hence the follicles were expressed while the patient was under a general anaesthetic. The plates were inoculated immediately and were then sealed by plasticine, care being taken that the surface of the culture medium was distinctly moist as 'dryness' inhibited growth. Thereafter, the plates were put in the dark and kept at room temperature, heat above 30° C. being adverse to growth.

In cases from which *B. granulosis* was recovered, a few minute 'dew drop' colonies appeared sometimes after a week but more often they were not seen until 15 days or even longer had elapsed. The colonies were subcultured several times to increase their vegetative properties and to insure the purity of growth. As the colonies grew older they became more opaque and passed through the stages of a dirty yellow to a brownish colour. As the plate originally used was repeatedly examined, even after the first colonies were seen, new minute ones continued to appear. Throughout, the colonies of the typical micro-organism were sticky, regular in outline and with a smooth surface.

A film stained by Gram's method showed the colony to consist of regular minute Gram-negative bacilli but in the older colonies their morphology became more pleomorphic. When examined by dark-ground illumination motility was a constant character.

Some colonies which were also composed of a Gram-negative minute bacillus which at first resembled those of *B. granulosis*, later became rough and umbonate; moreover, these did not ferment glucose.

If acidity was produced in glucose-peptone-water, further fermentation tests were carried out. The sugars were those mentioned in the paper by Finnoff and Thygeson to which reference has already been made, and prepared according to the formula of E. B. Tilden, namely, peptone-water containing 1 per cent. carbohydrate and 1 per cent. Andrade indicator.

The series consisted of 25 cases which from the clinical standpoint were undoubted examples of trachoma. Of these, no growth occurred in 5 cases. These belonged to the earlier part of the series and the fact that no micro-organisms grew on blood agar was probably due to the use of cocaine and to delay before inoculation of the plate.

From 13 cases colonies were obtained which at first had the cultural and morphological characteristics of *B. granulosis* but did not ferment glucose. Moreover, later the surface of these colonies became roughened.

From the remaining 7 cases, *Bacterium granulosis* was isolated. In 3 of these the properties of the micro-organism were the same in all respects as those of Noguchi's 'typical' form*; in 4, the micro-organisms were similar to Noguchi's 'atypical' forms.

The following table shows the fermentation reactions of the typical forms and demonstrates that they correspond exactly with Noguchi's results:—

Sugars	Noguchi	Case I	Case II	Case III
Glucose	+ + + +	+ + + +	+ + + +	+ + + +
Dextrose	+ + + +	+ + + +	+ + + +	+ + + +
Laevulose	+ + + +	+ + + +	− + + −	+ + + +
Mannose	+ + + +	+ + + +	+ + + +	+ +
Saccharose	+ + + +	+ +	+ + + +	+ + + +
Raffinose	—	—	—	—
Insulin	—	—	—	—
Galactose	+ + to + + + +	+ + + +	+ + + +	+ + + +
Maltose	+ + + +	+ +	+ + + +	+ + + +
Salicin	+ + + +	+	+ + + +	+
Xylose	+	+	+	+
Mannitol	+ + + +	+ + + +	+ + + +	+ + + +
Dextrine	+ + + +	+ + + +	+ +	+
Arabinose	+ to + + +	—	+ +	+ + +
Lactose	+ +	+ +	+ + +	+ + +
Dulcite	—	—	—	±
Sorbite	—	—	—	—
Inosite	—	—	—	—

It is necessary to indicate what constitutes an 'atypical' form. In morphology, staining reaction, motility, appearance of their growth on blood agar and action on glucose the atypical forms

* These three cases occurred late in the series and hence the remark made by one of us (S. S. M.) at the International Conference on Trachoma held in London in April, 1935, namely that *B. granulosis*, Noguchi, was not present in cases in the Glasgow area (*Brit. Jl. of Ophthal.*, Vol. XIX, p. 326, 1935).

correspond in every way to the 'typical' form; they differ, however, in their action on some of the other carbohydrates.

Much controversy has arisen as to the part played by *B. granulosis* in the aetiology of trachoma; moreover, other micro-organisms have been isolated and put forward as aetiological factors. Again, various authors have maintained that the condition is due to a virus, to diet deficiency and lastly that it is an expression of allergy.

With these views, however, this paper is not concerned. In our introductory remarks it was clearly indicated that considerable divergence existed in the results of those authors who had carried out work along lines similar to that of Noguchi and our investigation was undertaken merely to ascertain whether *B. granulosis* was present or not in cases of trachoma occurring in Glasgow.

We suggest that some of the discrepancies in the results obtained by various workers have been due to their not having allowed sufficient time for growth to have progressed to the extent of producing visible 'colonies' before pronouncing that the micro-organism in question was not present in the material examined. Again, some of these investigators may not have been conversant with the inhibiting action of cocaine on the growth of *B. granulosis*.

Lastly, the importance of immediate inoculation of the media, the keeping of the inoculated plates at room temperature and of maintaining a sufficiently moist surface should be emphasised.

Conclusion

An investigation has been carried out in order to ascertain whether *B. granulosis*, Noguchi, occurs in cases of trachoma in Glasgow. The results obtained, show that this organism is present in a proportion of these cases.

THE WATER-BINDING OF THE OPTIC NERVE AND OF ITS SHEATHS

BY

J. A. VAN HEUVEN *and* P. F. FISCHER

UTRECHT

AFTER our paper on the water-binding of the retina some interesting investigations have been published on the genesis of papill-oedema, and it seemed to be of some importance that similar researches on nervous tissues should be done. For these reasons we continued our experiments.

This time we took the optic nerve instead of the retina and worked very much after the same scheme. In doing so we investigated first the *capacity*, then the *intensity*, and finally the *form* of the water-binding.

Again we used ox-eyes, obtained as fresh as possible from the slaughter-house, which were brought to our laboratory still embedded in the orbits. The optic nerve and its sheaths were separated from all other tissues, after which the coat of sheaths was incised by means of a pair of scissors as far as the entrance of the optic nerve in the globe, and finally the optic nerve was separated from the globe in such a manner that the papilla remained attached to the optic nerve.

Now it appears to be of practical importance that as far as possible pieces of the optic nerve of the same size should be used. To get these we made use of a little cutting machine consisting of two glass tubes placed end to end and separated only by a small slit, sufficiently wide to allow a razor blade to be used. The glass tubes were mounted on a wooden base and contained a lumen which allowed the optic nerve to pass through.

By using this instrument it was possible to get pieces of optic nerve, all of which were 2 mm. long. As a rule, 10 to 15 of these small pieces were taken from each optic nerve.

These small pieces of optic nerve were weighed, then put in different liquids and then weighed again. From the difference in weight the absorption or the loss of water could be calculated in percentage.

The first liquid to be used was distilled water. From numerous experiments we learned that after one hour there was found an average increase in weight of 77 per cent.; after three hours the average increase in weight was 90 per cent., and after 24 hours 160 per cent. Thus it appears that the absorption in distilled water of the optic nerve is many times greater than that of the retina. Further experiments showed that this gain in weight of the optic nerve in water was, in fact, only dependent upon the absorption of water. Practically nothing was dissolved in the water, only after 24 hours the Cl reaction was slightly positive.

Looking at the optic nerve from an anatomical point of view we found that the further the small piece of optic nerve was taken away from the globe the greater the absorption of water appeared to be. Further numerous researches showed that in fresh condition the optic nerve contains more water near the globe than at some distance from the globe. As a matter of fact, the papilla of the optic nerve contains the greatest quantity of water. Though the differences are not great, they are found to be regular. About

half way the water percentage of the optic nerve is about 74 per cent.

If we take our small pieces of optic nerve and put them in acids instead of in water we get the following results: Schedule I shows the percentage of increase in weight, if we leave them in the liquid for three hours.

SCHEDULE I

	N	N/2	N/10	N/100	N/1000	N/10000
Hydrochloric acid ...	+19	+29	+47	+48	+56	+66
Sulphuric acid	+20	+24	+25	+37	+45	+54
Nitric acid	+18	+18	+36	+37	+42	+49
Acetic acid	+43	+44	+42	+36	+34	+33
Lactic acid	+82	+71	+65	+46	+35	+34
Citric acid	+86	+78	+67	+53	+48	+40

This schedule shows that the water absorption in acids is lower than that in distilled water. So the water absorption in acid solutions is retarded in comparison with that in pure water. During the first 12 hours this is the case in all acids. If the pieces of optic nerve are left in solutions of organic acids for over 12 hours the water absorption becomes greater than that in pure water. This means that in acids the water absorption is a slow process. Furthermore, it is clear from schedule I that in increasing concentrations the water absorption is retarded by mineral acids and promoted by organic acids. Equal normality does not mean equal water absorption.

We did not succeed in getting reliable figures representing the water absorption of small pieces of optic nerve in alkalies. The reason for this is clear if one looks at those pieces of optic nerve which have remained in an alkaline solution for more than one hour. A change has taken place in the pieces of optic nerve very similar to that which we found in the pieces of retina (*Brit. Jl. of Ophthal.*, p. 390, July, 1935). They show a lengthening on both ends, whilst the middle part remained either almost the same or even shrunk; after this they dissolved, sometimes in the form of long threads. This change is due to a water absorption which surpasses many times that in pure water. So we are safe in saying that the water absorption by the optic nerve is promoted intensively in alkalies. Furthermore, in alkaline solutions the same rule holds

good as in acids, *i.e.*, that equal concentration does not mean equal water absorption.

Schedule II shows the percentage of increase in weight of small pieces of optic nerve which remained for three hours in solutions of salts.

SCHEDULE II

	m	m/10	m/100	m/1000
Na-cyanide	+17	+39	+51	+69
Na-rhodanide	+30	+32	+35	+41
Na-nitrate	+19	+34	+38	+38
Na-iodide	+17	+16	+21	+28
Na-bromide	+21	+41	+55	+72
Na-tartrate	-1	+17	+30	+40
Na-phosphate	-3	+15	+27	+32
Na-sulphate	-10	+26	+41	+47
Na-citrate	-12	+15	+29	+39

Generally speaking, we may say that equimolar solutions show different effects. The water absorption is retarded in salt solutions; this retardation is more pronounced the stronger the solution.

If we take anelectrolytes instead of salts similar results are obtained.

From the behaviour of small pieces of optic nerve in solutions of acids, alkalies, salts and anelectrolytes may be concluded that their absorption of water must be looked upon as a real swelling, similar to what we have found in the retina.

Moreover, a comparison between the absorption of water by the retina and by the optic nerve shows that the capacity of the water-binding of the optic nerve is much greater than that of the retina.

Afterwards we tried to get some figures showing the *intensity* of the water-binding of the optic nerve. For this purpose we studied first the rapidity of the swelling. We made these experiments in the same way as we have done with the retina on a previous occasion in various bathing liquids. Here follow the figures for acetone and urea:

SCHEDULE III—CONCENTRATION OF ACETONE

Time in minutes	10 per cent.	20 per cent.	30 per cent.	40 per cent.	50 per cent.
1′	+10	+15	+17	+11	+6
5′	+12	+17	+18	+12	+5
10′	+12	+18	+19	+7	+5
60′	+29	+24	+26	+8	+4

A comparison of these with the corresponding figure of the retina shows that the optic nerve has the possibility of a quicker and more intensive swelling than the retina. In conditions of concentrations and time in which the retina loses water the optic nerve is still capable of absorbing water.

SCHEDULE IV—CONCENTRATION OF UREA

Time in minutes	10 per cent.	20 per cent.	30 per cent.	40 per cent.	50 per cent.
1′	+8	+13	+10	+10	+16
5′	+11	+9	+11	+12	+13
10′	+14	+12	+12	+17	+15
60′	+20	+24	+26	+34	+29

This shows that in urea in any condition of time and concentration the optic nerve absorbs water. Formerly we found that pieces of retina lose water when they are placed in solutions of urea for a short time or in a solution of high concentration. It is now demonstrated that the optic nerve has a greater tendency and potentiality to absorb water than the retina.

Furthermore, we have investigated the change of density in relation to the change in the water-percentage. We used the same method as before in our paper on the retina. This time, however, mercury was used as a liquid, as in both toluol and xylol the weight of the little pieces of optic nerve was changed.

Fig. 1 shows the change in specific weight in relation to the percentage of water. If the percentage of water increases the density is changed regularly, but slowly in comparison with the retina. Both in the retina and in the optic nerve the water-binding is the same in all stages, there are only quantitative differences.

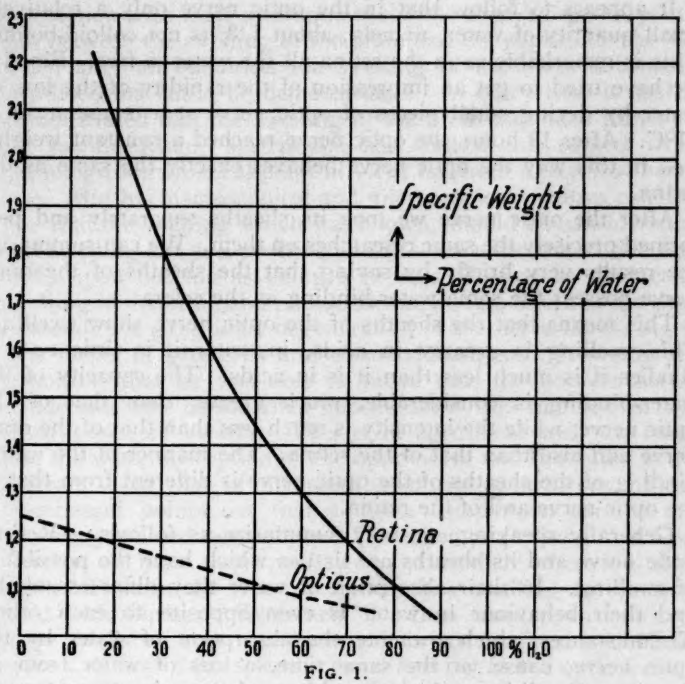


FIG. 1.

If the percentage of water decreases, the specific weight increases; if the percentage of water increases we get a relative diminution of the volume. This is smaller in the case of the optic nerve than in the retina.

Schedule V shows the rapidity of the loss of water in solutions of acetone of 75 per cent. and of 100 per cent.

SCHEDULE V

Time in minutes	75 per cent. Acetone		100 per cent. Acetone	
	Loss of water	Percentage of water	Loss of water	Percentage of water
1′	−2	71	−8	76
5′	−1	70	−3	68
10′	−3	68	−15	72
60′	−5	73	−22	73

It appears to follow that in the optic nerve only a relatively small quantity of water, namely, about 1/3, is not colloid-bound. This is remarkable as in the retina all the water is free. Finally, we have tried to get an impression of the rapidity of the loss of water by drying small pieces of optic nerve at a temperature of 37°C. After 19 hours the optic nerve reached a constant weight, *i.e.*, in this way the optic nerve behaves exactly the same as the retina.

After the optic nerve we took its sheaths separately and performed precisely the same researches on them. We can summarize the results very briefly by saying that the sheaths of the optic nerve possess the same water-binding as the sclera.

This means that the sheaths of the optic nerve show swelling. This swelling is greatest in acids, in water it is little and in alkalies it is much less than it is in acids. The capacity of the water-binding is considerable, much greater than that of the optic nerve, while the intensity is much less than that of the optic nerve and also than that of the retina. The manner of the water-binding of the sheaths of the optic nerve is different from that of the optic nerve and of the retina.

Generally speaking, we may summarize as follows:—Retina, optic nerve and its sheaths are tissues which have the possibility of swelling. In their absorption of water they differ essentially and their behaviour in water is even opposite to each other. Circumstances which promote the absorption of water by the optic nerve, cause, at the same time, a loss of water from its sheaths. Conditions which are able to change the percentage of water of the optic nerve are without significance for the retina, and *vice versa*. This different behaviour of tissues which are so closely situated next to each other cannot remain without clinical significance and without clinical manifestations.

Different authors as a matter of fact have already assigned to the power of water-binding of the optic nerve an important rôle: in a discussion during the year 1930, Marchesani stated that according to researches of Spatz and himself, atrophia cerebri senilis was caused by a decrease in the power of water-binding of the brain. The optic nerve being part of the brain has to share these changes.

Marchesani made the following demonstration. He took a cat and injected in its right carotid artery pure water and in its left carotid artery a solution of 10 per cent. NaCl, both under the same pressure. The right part of the brain and the papilla of the right eye swelled, while at the same time the left part of the brain and the papilla of the left eye shrunk. The latter is shown by an excavation of the left papilla.

In 1934 Marchesani again stated that in 120 brains, still in

connection with their own optic nerves he always found a parallelism between the swelling of the brain and the papilloedema. Only when after an operation the swelling of the brain subsided no papilloedema could be demonstrated. In every case it could be demonstrated that the swelling of the brain had been previous to the papilloedema. Marchesani has studied this subject from a pathologico-anatomical point of view, not from a physico-chemical one. But his macroscopical and microscopical pictures convinced him that the swelling which was shown, should be considered as a real swelling not as a swelling in the sense of oedema. He rather looks upon oedema as being a process of opposite character to swelling of the brain and papilloedema, no free liquid in the tissues is found, but ganglionic degeneration of the nerve fibres, big and pale nuclei of glia cells and clasmatodendrosis. It seems to be important that he was able to prove that the borders between ectodermal and mesodermal tissues show this increase in water absorption very clearly. This is in agreement with us, namely that the water absorption of the optic nerve and the retina is quite different from that of the sheaths of the optic nerve. Finally Marchesani points out that a direct comparison can be made between papilloedema and the " Zysternenverquellung " of Spatz. Namely, the brain tissue swells everywhere there where it is able to swell. Thus in the first place there is a swelling in the spaces filled with liquor (" Zysternen "). In a similar way the optic nerve swells on every spot where it is not confined by inflexible tissue *i.e.*, not in the sclerotic canal and not in the optic foramen, but it swells protruding into the globe.

These anatomical and histological observations of Marchesani are important, but they lack colloid-chemical proof. Discussing this we may say that many years ago M. H. Fischer and Marion Hook had already proved that the absorption of water by nervous tissues represents a real swelling. From our papers it looks as if the optic nerve, its sheaths and the retina are also tissues which have the possibility of swelling. Thus a theory of papilloedema founded upon swelling of brain and optic nerve, as has been given by Marchesani and Spatz, might be possible. Only one has to be on the outlook for facts which would directly contradict such a theory.

There is another experiment which is in favour of Marchesani's views though it has not been mentioned by him. In fresh cases of papilloedema no signs of inflammation are found. Neither is a real oedema present *i.e.*, in no case is water found, the tissues are swollen but no water can be traced between the tissues.

Furthermore it is not possible to obtain a papilloedema by increasing the intra-cranial pressure, as lately has been proved very clearly by Lindberg. He took rabbits and kept their intra-cranial

pressure for 3 hours at 40 mm. Hg; then until their death he increased the pressure to 60-80 mm. Hg. By doing so he did not get a papilloedema though the veins were wider and the arteries narrower.

On the other hand it was found by Bauermann and Sobansky that the pressure in the veins of the retina is caused by the intracranial pressure. Moreover that papilloedema can be found only when there is an abnormal relation between the arterial and venous pressure of the vessels of the retina. In this way Sobansky succeeded in getting temporarily a papilloedema in a dog. By stopping in a dog the venous outlet of the optic nerve without interfering with the arterial supply *i.e.*, by changing the relation in pressure in the vessels of the retina without changing the intra-cranial pressure he was able to observe after 3 minutes the beginning of a hyperaemia of the papilla and after 6 more minutes a swelling of the papilla. Are there now any relations between these changes in pressure and the absorption of water ?

It is shown that in every case changes in pressure are the *primum movens*, that rather considerable changes in pressure are necessary to cause some effect, and that the change in pressure must last for some time before a papilloedema is obtained.

Sobansky found that the changes in pressure represented an approach of the different pressures to each other. Thus they lead to a stasis of venous blood and consequently to a retardation of the metabolism. This causes different changes and the result might be that there is an increase in the amount of swelling of the tissues. Such a process takes time. Therefore it is not seen immediately at the beginning of the experiment or the disease. As long as there is only swelling of no excessive degree the function remains relatively good. As is the case in all swelling phenomena these processes are reversible, so it appears to be quite possible to connect and complete the theories of Marchesani and of Bauermann and Sobansky with each other. The latter is not able to explain why changes in pressure lead to a choked disc, and the first is not capable of explaining the relations between changes in pressure and papilloedema. Nevertheless Marchesani's theory is the more important of the two. For, having first stated that the condition of the part of the optic nerve which can be seen by the ophthalmoscope depends on its degree of swelling, it is easy to explain the opposite condition *i.e.*, an atrophy of the papilla by decrease of the swelling, from the same point of view. Marchesani indeed has already discussed the possibility that those cases which have been described by Thiel, and in which pale discs containing an excavation were seen, might have been caused not by atrophy but by a change in the water percentage of the tissues.

Various other examples might be given, illustrating relations between colloid-chemical researches and clinical problems *e.g.*, atrophies by pressure, condition of the disc in the Foster-Kennedy syndrome etc.

Summary

Researches are made on the water-binding of the optic nerve and on that of its sheaths, and it is proved that these tissues have the possibility of swelling. The capacity and intensity of the water-binding of the optic nerve is much greater than that of the retina. Moreover the water-binding of the optic nerve is of a different type from that of the retina. The conditions of water-binding are the same in the sheaths of the optic nerve as in the sclera. Furthermore the clinical significance of the water-binding of the optic nerve and its sheaths is discussed and some remarks are made on the theories of the genesis of papilloedema.

REFERENCES

Bauermann —*Ber. d. deutsch. ophthal. Ges.*, p. 53, 1925.
F. P. Fischer —*Arch. f. Augenheilk.*, Vol. CIII, p. 1, 1930.
J. A. van Heuven and F. P. Fischer.—*Brit. Jl. of Ophthal.*, Vol. XIX, p. 390, 1935.
M. O. Hooker and M. F. Fischer.—*Kolloid-Zeitschr.*, Vol. X, p. 283, 1912.
Lauber, J.—*Wien. klin. Wochenschr.*, Vol. LI, p. 1, 1934.
Lindberg, J. G.—*Finska Läk. sällsk.*, Vol. LXXVI, p 694, 1934.
Marchesani, O.—*Ber. d. deutsch. ophthal. Ges.*, p. 157, 1930; p. 356, 1934.
Sobanski.—*Kl. Oscna*, p. 360, 1933.
Thiel.—*Ber. d. deutsch. ophthal. Ges.*, p. 133, 1930.

ON THE SCIENTIFIC AND PRACTICAL VALUE OF IONIZATION IN OPHTHALMOLOGY. RECENT ADVANCES AND RESEARCHES

BY

DR. GUSTAV ERLANGER

NEW YORK

IONIZATION—the introduction of drugs and electrolytes into the tissues of the human body or into those of the body of animals with the aid of a weak direct current—plays an important rôle in recent medical literature. This method has been studied especially in ophthalmology. And here its great therapeutic effect may be explained either by experiment or by clinical experience.

The pharmaco-dynamic effect of the current plus the electrolyte consists in a sudden alteration of the permeability of the tissues and with it also of the surface tension. We fix the electrolytes somewhat longer to the cells, we are able to irritate the sympathetic

and the parasympathetic nerves with this method in a better way and we may change the circulation in a sense which is favourable to the restoration of normal or nearly normal functions.

Another feature in adopting this method systematically is the possibility of introducing, especially into the tissues of the eye, in a new way the so-called urticariogenous group, such as histamine, acetylcholine and so on. This method has a specific effect on the vessels and with ionization these substances can be introduced directly into the tissues, whilst by injecting them subcutaneously there is no guarantee that they come in a sufficient quantity to the site of the lesion.

The technique of ionization nowadays is very simple. We use an apparatus called Sanion, which contains four pocket batteries, an exact milliampèremeter, a socket and a switch. The wires go to a hand-electrode which can be provided either with a capsule or with another wire to be connected with a metal tube. The capsule must be filled up with cotton-wool soaked in fluid. The tube must be fixed at the top with a piece of cotton-wool, which one may form corresponding to the size of the ulcer or the focus one is going to treat. The switch is put to the first position. If the patient touches the eye with the filled and wet capsule the current is closed. To have more intensity of current the switch has to be turned to position two or position three. To avoid sensations, let the capsule be brought near the eye from the touched skin.

In using the tube the physician takes it into his hand and puts it on the cornea or the sclera or the lids, after having placed the hand-electrode in the hand of the patient.

We need only small intensities of current. To obtain therapeutic results when using the capsule, intensities from 1-2·5 milliampères, time 3, 5 or 10 minutes when using the tube; intensities from 0·5-2 milliampères, time 0·5-2 minutes are sufficient.

It may be mentioned here that in the past year we used activated solutions with the best effect. The common solutions of electrolytes may be used, but a better effect is afforded by the activated Sanion-solutions, because they are provided with catalysators. These catalysators give no trouble at all to the effect of the ionization and are proved to increase the therapeutic effect in the cells and in the tissues.

To prove the efficacy of ionization we mention some of the experiments on the eyes of rabbits and of guinea-pigs. In experimental work we use the dose of 3 milliampères for 3 minutes. For instance, can we produce from the *positive* pole with adrenaline (1 : 1,000) a mydriasis of about 11 mm., which lasts several hours. When we take a solution of adrenaline 1 : 1,000,000 we may produce this mydriasis 45 minutes after the ionization.

At any rate, we have an effect on the pupil with even highly diluted solutions with this method.

The ionization has not only an effect in the tissues in which the drugs are migrating; it changes, also, the whole circulation, as can be proved by measuring the intra-ocular pressure. In general, there is an increase of the intra-ocular pressure directly after ionization, even after introducing pilocarpine and eserine. This increase of the intra-ocular pressure is followed by a decrease, which sometimes assumes a diminishing of the pressure by 5-8 mm. Hg. Another proof that the circulation of the eye is influenced: if we inject intravenously 5 c.c. of a 5 per cent. solution of fluorescein (into the vein of a rabbit's ear) and we perform ionization five minutes later, for instance, of sulphate of zinc on the cornea, the whole anterior chamber is filled up with the stain after some minutes. Normally, there is only a thin line of fluorescein from the upper part of the pupil down to the bottom of the anterior chamber, the line of Ehrlich.

That there is also a specific effect on certain tissues can be proved by the following experiment: In introducing chloride of barium (1 per cent.) from the *positive* pole to the eye of a guinea-pig, we always produce a cataract, beginning immediately after the ionization, and forming within 48 hours a total cataract, whilst the cornea remains clear. The effect of the barium can only be overwhelmed by iron when introduced together with the barium by ionization. Fig. 1 shows clearly the cataract. The section

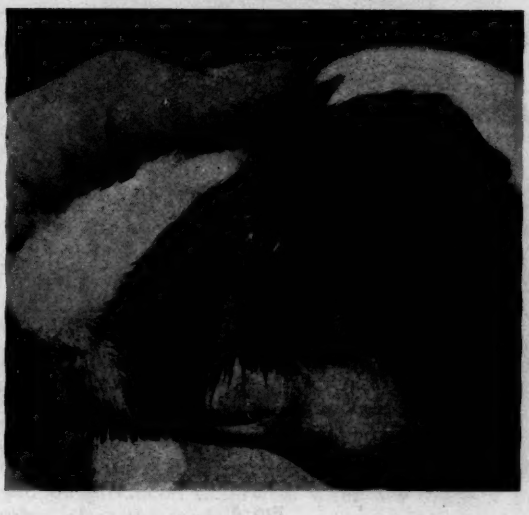


FIG. 1.

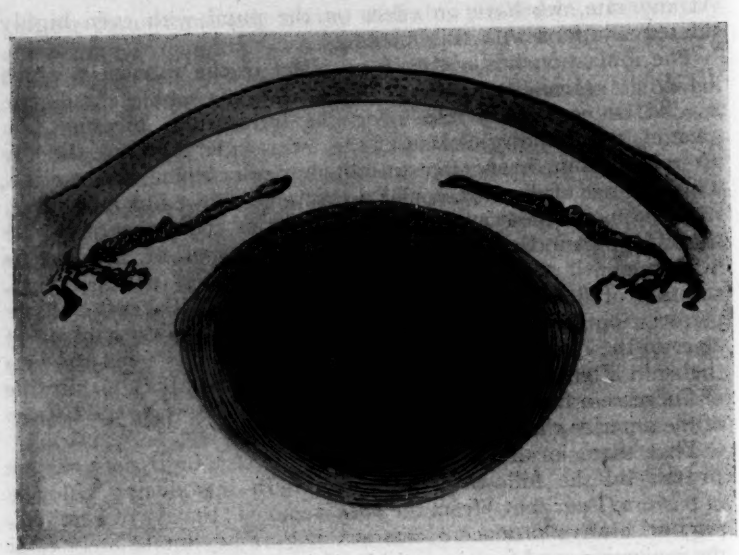


FIG. 2.

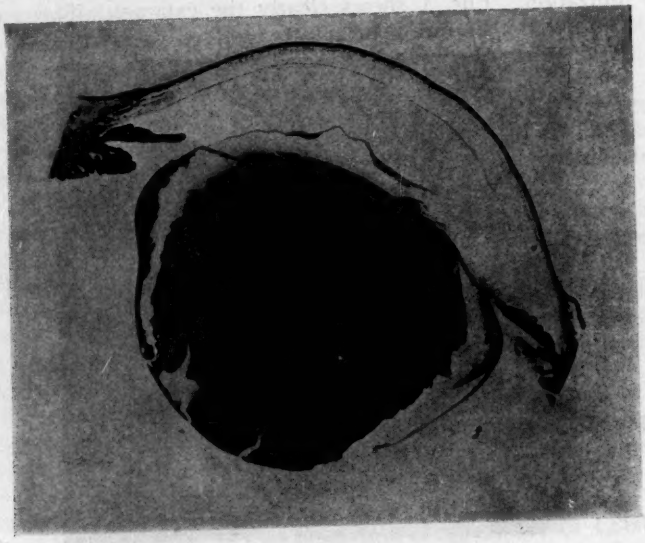


FIG. 3.

(Fig. 2) shows a normal eye of the guinea-pig, whilst the section (Fig. 3) shows the homogeneous change of the lens, the total cataract.

With this experiment we could study the problem of avoiding a cataract. It seems that thyroxin subcutaneously injected (1 c.c.) before the ionization with barium, has a certain influence in preventing cataract. Some sections of eyes in this experiment show an enormous dilatation of the vessels of the choroid which one never sees in any other condition. So that we may say that by changing the circulation with the aid of thyroxin, which we bring also by ionization in a larger quantity into the eye, the

FIG. 4.

oxidation of the eye is augmented, so that the barium is unable to alter the lens. All these experiments must, of necessity, lead to a new study of the question of cataract.

If we introduce into the eye of a rabbit a solution of hydrogen peroxide (1 per cent.) from the *positive* pole, we may produce, in some cases, a real megalocornea, with a diameter of the cornea of 12 mm. instead of 8 mm. with a low pressure of about 8 mm. after some 14 or 20 days (Fig. 4). In one case we saw after ionization bubbles in the anterior chamber. Bubbles of oxygen? These bubbles could be produced otherwise when we made a puncture of the chamber after the ionization.

All these above mentioned experiments prove that the method of ionization is a method of great pharmaco-dynamic effect, worthy of being studied and, when modified, also of great therapeutic value.

For 15 years we had the opportunity of seeing many dogs with severe diseases of the eye. In applying ionization by different methods to these diseases we could study its great therapeutic value. There is no doubt at all that this method is superior to any other method up to the present. We saw cases treated with all known means, and without any avail. The very moment of using ionization the pathological picture changed. Let us see, for instance, such a picture as keratitis in distemper with a perforation and prolapse of the iris (see Fig. 5). The conjunctiva

FIG. 5. FIG. 6.

is suppurating, after some days the cornea is affected, becoming opaque; there is loss of epithelium, an ulcer is forming and suddenly the ulcer perforates. The iris is attached to the cornea. A certain vascularization is evident after some time. The cornea remains cloudy, the iris is attached to the cornea in spite of the use of atropine. We begin treatment after anaesthesia with cocaine 2 per cent. or pantocain 1 per cent., with 0·25 per cent. sulphate of zinc with some drops of adrenaline (1 : 1,000) 1 milliampère 1-2 minutes. The next day there is a change in the appearance of the eye. The vessels of the livid vascularization are spreading in the direction of the hole in the cornea. The second treatment consists in applying calcium chloride 0·5 per cent. with drops of adrenaline to the whole cornea. So we change calcium with zinc, always with adrenaline. At the end of a week we have quite another system of vascularization, the cornea is transparent, the iris has been drawn back from the cornea and we have a round pupil

again (see Fig. 6). The vascularization consists of a few vessels only, which provide the cornea with better nutrition, for the vessels look quite red instead of the livid colour of the former vascularization. With the aid of zinc we secure the progress of the growth of epithelium, with adrenaline we exercise an irresistible traction on the iris, with the frequent application of calcium we sensitize the sympathetic nerve so that we get an active hyperaemia instead of the passive hyperaemia which makes only a congestion of vessels and produces that livid ring of vascularization. It is impressive to observe how only a few vessels are necessary for the healing of a big ulcer, so that the cornea again becomes transparent. We see here the confirmation of a real biological law: minute irritation leads to an ideal restoration of the severely damaged cornea. This restoration is even possible in spite of the continuing suppurative exudation of the conjunctiva. There is no possibility any more of infection of the cornea, so great is the effect of the method on the resistance of the tissues. All the cases were treated without any bandage.

From an experimental point of view we tried to treat some cases of staphylomata of the eye, which we saw by chance. Such dogs are otherwise killed by the veterinary surgeon. It was astonishing to observe the vascularization migrate to the top of the staphyloma. The cornea cleared up and at the end of a week a prolapse of the iris appeared. After some 10 days this disappeared and a round globe developed out of the former staphyloma (see Figs. 7 and 8). Such a therapeutic effect is really convincing and no scepticism is any more permissible.

With the above mentioned method we could in every acute case of perforated cornea bring back into the anterior chamber the prolapsed iris, in most of the cases even with a free moving pupil. But also in old cases of iris prolapse we had a good result. We mention the case of a dachshund which had a subconjunctival prolapse of the whole of the upper part of the iris. After 10 treatments the iris was completely in the anterior chamber. The cornea, which had a hunch in the upper part, became quite clear and normally curved, the irritation of the eye which had existed several months had ceased altogether. One year later the eye was in the same good condition.

From all these experiences, experimental and clinical observations, the idea developed to study such a method systematically in the treatment of various diseases and pathological changes in the eyes of human beings. And also in this field the method of ionization brought new and interesting results. There existed already a good deal of scientific literature, especially of an ophthalmological character, with reports of interesting therapeutic results. We mention the papers of Wirtz, 1908, and Schnyder, 1919. These

FIG. 7.

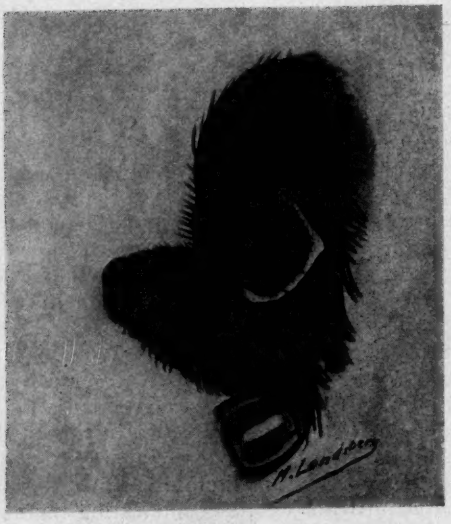


FIG. 8.

authors confined their iontophoretic work to but a few diseases. The results were worth taking into account, but scepticism arose and the method was only performed by a few ophthalmologists. Bos published a larger paper on ionization in 1929. He reported on the results in treating ulcers of the cornea, further on the result with ionization of atropine in cases of iritis. His results were better than with the usual methods. His chief, Dr. Rochat, the director of the eye clinic of the University Groningen, gave new communications on this subject in the International Congress of Ophthalmology in Amsterdam, 1929. A condensation of my work was given by me in Amsterdam, and in 1932 in a pamphlet in the *"Jahreskurse für ärztliche Fortbildung,"* J. F. Lehmanns, Verlag, München.

But even since that time the systematic ionization of some new drugs led to the extended application of the method in the diseases and changes of the posterior part of the eye—the results in these diseases are of the most remarkable significance.

Indications.—Besides the usual therapy the conjunctiva may be treated in cases of acute inflammation with iontophoretic introduction to the everted lids. Zinc sulphate is to be applied to the tube, 0·25 per cent., 1-2 milliampère, 1-2 minutes.

In chronic cases the application of a mixture of calcium chloride and zinc sulphate is of better avail. The dose is the same as before. Calcium chloride 0·5 per cent., zinc sulphate 0·25 per cent. *and partes.*

The follicular conjunctivitis reacts very well and very quickly to the application of calcium chloride 0·5-0·25 per cent., 1 milliampère, 2-3 minutes. Nümi, a Japanese author, gives iontophoresis with a solution of 25 per cent. of calcium chloride. He reports very good results (see *Klin. Monatsbl. für Augenheilk.*, April, 1935).

The different kinds of catarrhal ulcers of the cornea are a proof of the strong influence of the ionization. With the tube one touches the ulcers and the surroundings. Zinc sulphate with one or two drops of adrenaline, 1-1·5 milliampère, 1-2 minutes. Sometimes a small vascularization is developing which disappears shortly afterwards. After some ionizations with zinc, calcium with two drops of adrenaline is to be applied.

As to the ulcus serpens corneae we have seen very good results with ionization of optochin 1 : 600. Dose 1-1·5 milliampère, 1-2 minutes. In severe cases it is necessary to treat the ulcer twice a day. If optochin is lacking one can use a mixture of zinc sulphate with quinine chloride, to which solution is added one drop of adrenaline. Zinc 1/400, quinine 1/200 *ana partes*. Dose the same as above.

The scars after ionization with this means are much more transparent than those obtained with any other method. Bos, Arruga and Malkin report similar good results. Arruga alleges that the bactericidal effect of optochin, for instance, is due to the fact of optochin migrating into the depths of the tissue, an opinion which corresponds to all of our reported observations.

The effect of epithelialisation of zinc is shown best in cases of herpes of cornea. After some treatments with zinc every day, the loss of epithelium diminishes and the final vision is much better than with every other method. In herpes and all sorts of neurotic disturbances only one thing is important; no large dose, 0.5-1 milliampère, 0·5-1·5 minutes. When four or five treatments with zinc have been performed, calcium should be taken, also with the capsule 3-5 minutes, 1 milliampère.

The rare keratitis disciformis can be wholly cured with the ionization of zinc in a very short time. In a paper in 1922 I stated that I could heal such a keratitis in less than three weeks, with nearly normal vision. Fuchs says in his textbook of ophthalmology about keratitis disciformis: "The course is protracted, as it takes one or more months for the eye to become free from congestion and for the infiltrate to be transformed into a corneal opacity which is permanent."

The post-operative infections of the flap in Elliot's operation can be cured, sometimes in one sitting, by ionization with zinc sulphate 0·5 per cent. We use the tube, 1·5 milliampères, 2 minutes.

Very impressive is the therapeutic effect of the method in all cases which last months and months without any reaction to other therapeutic methods. We have seen sufficient cases to be convinced of the method. Let us describe only a few.

A patient was treated more than three months daily in a clinic on account of keratitis ulcerosa. The cornea was sowed over with a multitude of infiltrates when we saw the patient first. After some ionizations with zinc the cornea was free of infiltrates, no irritation at all and the vision was normal. No further recurrence occurred.

Another patient, a bus driver, suffered six months from a recurrent keratitis with irritation of the iris. After three ionizations with zinc and adrenaline we did not see the patient for three weeks. He came back with a normal eye and told us that he had driven his open car in bad weather directly after the last treatment, because he had no trouble at all. No recurrence.

Two years ago a man came to us. He had been treated in a clinic six weeks. His right eye was blind from old kerato-iritis. His left eye suffered from the same disease. An operation (iridectomy) was indicated as the last remedy. The vision of the highly inflamed eye was 1/30. We performed ionization with zinc,

calcium and adrenaline daily. After the second treatment the vision arose to 1/10. After 14 days the eye was not any more inflamed, the vision was 1/4, the patient had no trouble with his eye and began to work. To avoid recurrences we treated him with calcium once a week for some months. The patient has been well for two years.

Trachomatous conjunctivitis was, in former times, treated by ionization with a mixture of zinc and copper. Now we favour a mixture of calcium chloride 1 : 300, zinc sulphate 1 : 400, quinine chloride 1 : 500 *ana partes*, which solution is brought to the everted conjunctiva, 1-1·5 milliampères, 2-3 minutes, every second day. The same solution is applied to the cornea with the tube, but only a small dose, 0·5 milliampère, 1 minute. Very effective is also the solution applied to the closed eye with the capsule 2-3 milliampères, 3-4 minutes.

In acute cases a Japanese author recommends the ionization with a 25 per cent. solution of calcium. Nümi uses this solution during 10-15 minutes, 2-4 milliampères. He has seen development of scars after 1-2 weeks. Amongst 55 cases he could cure wholly 39 cases and 14 cases showed improvement (Report in the *Klin. Monatsbl. für Augenheilk.*, p. 561, 1935).

In 1925 we saw a patient with a large ulcer of the cornea due to a trachomatous infection. This ulcer rose above the level of the cornea and could not be influenced by any of the usual means. We declined cauterisation, proposed by a consultative colleague. The very nervous patient finally gave his permission for the iontophoretic treatment. After three treatments with zinc the ulcer began to diminish in size and colour. After some eight days, instead of the ulcer there was to be seen a thin macula. Vision improved from hand movements to 1/3-1/2. A year later on, the other eye was perforated as the consequence of a similar ulcer and became blind, this eye not having been treated by ionization, as our representative did not know the method.

An interesting confirmation of the excellent effect of ionization is a report of Korrenjewicz, Kiew. He treated one eye with a pannus trachomatosus by ionization, the other with the same pannus as usual. In the eye treated with ionization the inflammation ceased surely and rapidly.

Episcleritis and scleritis react well to the ionization with histamine 1 : 10.000 or acetylcholin 1 : 400. The tube is put to the boss, dose 1 milliampère, 1-2 minutes. Treatment every day or every second day.

Infiltrates in tuberculous keratitis can be treated by ionization with calcium chloride 1 : 300. We use the capsule, 1·5 milliampères, 3 minutes twice a week for a very long time. A specific treatment is no hindrance. The resorption of the exudates is hastened with

this proceeding. (Report in the *Zeit. für Augenheilk.*, Berlin, April, 1935. "Especially impressive are the results in myopic opacities of the vitreous and even in tuberculous haemorrhages.")

A good field for ionization is the inflamed iris. We cite Rochat, who gave a lecture on ionization with atropine at the International Congress of Ophthalmology, Amsterdam, 1929. Atropine 1 : 1,000 is introduced into the inflamed iris, not more than 1·5 milliampères, not more than 2 minutes. No intoxication is possible. The treatment can be repeated every day or every second day without any danger. Rochat asserts that this treatment saved the patients from surgical interference.

We apply to the iris calcium with adrenaline, with the tube, 1 milliampère, 1·5-2 minutes, and in many cases after the treatment get the iris bloodless. When we then instil atropine in the common 1 per cent. solution, we strengthen the effect of the atropine. Sometimes we go with the tube only to the limbus and treat especially those spots where synechiae are present, because the adrenaline can sever the attached iris from the lens.

In subacute and even *chronic iritis* one should try at any rate an ionization with calcium first, then with calcium-adrenaline. The first treatment has to be done with the tube, then we combine the tube treatment with a treatment with the capsule. Tube: 1-2 minutes, 1 milliampère; capsule 5-10 minutes, 1-2 milliampères. It is only this method which in some old desperate cases changed the inflammation all of a sudden and produced sometimes surprisingly good vision.

Here let us remember the possibility of intensifying the effect of subcutaneous or intra-muscular injections of specific or non-specific means by making five minutes later on the ionization. In all the diseases of the iris it is often necessary to make such injections. Never forget to make the ionization after the injection.

The influence of silver-nitrate 0·2 per cent. from the positive pole, found experimentally in the rabbit's eye, could be proved. The tension can be diminished after a short time. An acute glaucoma in a dog's eye could be cured in one sitting. The intra-ocular pressure was diminished from 80 mm. Hg to 25 mm. Hg. Dose: 2 milliampères for 2 minutes.

Some human eyes with absolute glaucoma were treated in the same manner, with the result that the planned enucleation could be avoided.

In chronic glaucoma we treat the eyes in order to improve the circulation by ionization with a solution of calcium chloride 1/300, capsule-electrode, 5 minutes, 1·5 milliampères. Our friend, Professor Itoh, from the University clinic for eye diseases in Chiba, Japan, told us that he obtained good results with ionization of pilocarpine 1 : 1,000 in chronic glaucoma. He told us that he

had observel that not the pressure changed as much but that the vision and the field of vision remained at the same condition as in beginning the treatment.

As to the question of ionization-treatment in cataract the practical part of this question can only be solved in common with the work of many ophthalmologists who are adopting this method. It seems to us that this treatment must consist in improving the oxidation of the lens, which may be done directly and indirectly. Indirectly by all methods which improve the whole circulation, directly by applying with the aid of the ionization means which are improving the circulation of the whole eye, as eserine salicylate in a solution of calcium (calcium chloride 1/300 with 1-2 drops of eserine salicylate (0·5 per cent.). We use the capsule, 5-10 minutes, 1-2 milliampères once a week. We dare not say that this method is able to prevent the final progress, because a very large series of cases must be treated in order to gain a distinct impression of its effect. But of all experience we say the study of treatment of cataract must be done at any rate. We state from the experiment on the guinea-pig's eye that it is possible even in nearly totally non-transparent opacities of the lens to clear up these opacities under certain circumstances. And this observation leads to the conclusion that one day the medical treatment of the cataract will be possible.

The study and the systematic application of the ionization revealed its great and very often astonishing effect in treating many of the acute and chronic diseases of the eye with following improvement of inflammation and with it of vision, but the therapeutic effect of the method on the posterior part of the eye was a long time without producing satisfactory results.

The possibility of exercising a therapeutic effect with ionization on the posterior part of the eye, on the vitreous, the choroid and retina and even on the optic nerve, was given with the introduction of the so-called "urticariogenous" group. These systematically applied by ionization are histamine, acetylcholin, doryl, dionin. The effect of these is different only in qualitative aspect. For most of the treated cases the application of histamine, acetylcholin, dionin proved to be satisfactory. In application of acetylcholin we have worked out a new technique.

All these substances are formed in the tissue itself. Feldberg and Schilf have called them " Gewebshormone " (hormones of the tissue). These substances may be produced in the tissue by different stimuli. All these substances are working upon the vessels, dilating them and relieving spasm, producing arterial hyperaemia, intensifying the velocity of the blood corpuscles and increasing the flow from the veins. Histamine, besides, produces a marked oedema.

If we introduce such substances by ionization into the tissue of the eye we imitate a natural process, but with the difference that we may dose it to a therapeutic effect. This therapeutic effect may be got by the circumstance that ionization fixes the substance somewhat longer to the tissue, so that a prolonged effect is produced. The effect on the retina and choroid is, perhaps, not a direct one. There may chiefly be reflectory effects, if we dilate the vessels of the conjunctiva and those of the orbit. Another idea is possible:

By introducing these substances into the sclera we sensitize the ganglion cells, which are to be found in the sclera, as Dr. Goldstein, New York, told me, and as has been communicated by Dr. Ernyei (Hungarian Ophthalmological Society, February 16, 1935). These cells are of sympathetic character, so that the ionized substances have a direct effect on the sympathetic and with it are always producing a better hyperaemia than the usually applied means.

Very interesting are the communications of Ruhmann, who could show that the ionization of acetylcholin produces the same micro-capillary picture as an energetic massage. We cannot massage energetically the eye ball, but we are able to make a powerful dilatation of the vessels by the ionization of the sclera with acetylcholin.

The technique for the ionization of histamine is very simple. We anaesthetise the eye with some drops of 2 per cent. cocaine or of 1 per cent. pantocain. Then we use the tube with cotton-wool and a solution of histamine (1 : 10,000) and put the tube to the sclera far from the limbus, ordering the patient to look in the opposite direction. It is sufficient to treat the sclera at the temporal and at the nasal side. Dose: 1-1·5 milliampères, 1-2 minutes each site. The treatment can be repeated every day, in severe cases twice a day. If there is any reaction every second or third day. If there is extensive oedema warn the patient, because it lasts sometimes 5-10 hours. If there is a little haemorrhage in the conjunctiva, it does not disturb the therapeutic reaction. Dionin (1 : 500) has similar reactions, but histamine is of surer effect.

The technique of acetylcholin-ionization (1 : 200) has been modified. We can use the capsule, filled up with cotton-wool and with the solution of acetylcholin; dose: 2 milliampères, 5-10 minutes. To mobilize acetylcholin in the tissue itself and to increase its effect when introduced into the tissue by ionizaton it has proved best to use the capsule with a solution of calcium chloride (1 : 300) to which are added two drops of eserine salicylate (0·5 per cent.); dose: 1-2 milliampères, 5-8 minutes, from the positive pole. Eserine has the quality even in the highest dilutions of preventing the splitting up of acetylcholin by destroying the

enterase (Feldberg). After having introduced the eserine with the capsule we take the tube with cotton-wool and acetylcholin (1 : 200) and treat the sclera, the temporal and the nasal side, as far as possible from the limbus. Dose: 1-2 milliampères, 1-1·5 minutes each side. This treatment must be done in the beginning every day, then three times a week and after having got a result, once every week for a long time.

Generally, it may be said that in every kind of choroido-retinitis, be it of arteriosclerotic, diabetic or any other constitutional origin, the method should be systematically carried out. It is always possible to produce a better circulation, perhaps of the effect of the activated means, an effect, which consists first in

FIG. 9.

FIG. 10.

relieving the spasm of the small arteries. At the very moment of influencing the spasm the circulation is improving and the retina is functioning better. One of the most remarkable signs of improvement is the disappearing of central scotomatata for colours. The central vision is improving gradually, as well for distance as for near. That also in the course of such a treatment exudates in the retina are resorbed, can be proved by pictures of the fundus, photographed by the Zeiss-Nordenson camera. (See Figs. 9 and 10.) It was a case of arteriosclerotic retinitis. In the macular region a big pigmented patch was to be seen. The vessels from the optic nerve to the macular region disappeared behind an exudate. The vision was 1/25-1/20. Large paramacular scotomata could be demonstrated. After a treatment of more than two months the picture has changed in every way. You see, instead of the patch, a small haemorrhage, the previously curved vessels are stretched and well filled up, the veins and the arteries which had disappeared behind the exudate, are distinctly to be seen. The vision has improved to 1/3, with a weak convex-cylinder even to 1/2.

To illustrate the therapeutic effect of the new technique let us cite some other interesting cases:—

Thrombosis of the central vein. After 10 treatments improvement of vision from 1/50-1/35 to 5/35. Man, aged 63 years. Deep excavation, optic nerve temporal paleness. Vision from 1/35-1/25 with −8 0 D. Sph. excentrically seen, within four weeks to 5/20. Reading was improved from recognising big letters to recognising the smallest letters. Lady, aged 35 years, burning of the macula, after wandering over a glacier without protective glasses. Vision improved from 1/3 to 5/5, for near from Snellen 3·5 to Snellen 1·5 within 10 days (treatment every day). A central scotoma for red and green disappeared after the second treatment. High myopia is without any doubt a good field for this treatment. There are remarkable improvements of vision for distance and near.

That in all these cases the constitutional treatment must be taken in consideration is natural. At home, the patients have to instil every night a drop of pilocarpine (0·01/20) for months and months.

In cases of retinitis pigmentosa we combine the ionization with a diathermic treatment before, 10-15 minutes, 200-500 milliampères. There is no doubt that in some cases a therapeutic reaction is possible. Central scotomata for colours disappear. In one case we saw the field of vision enlarging in a high degree, instead of the narrow field of vision an annular scotoma had developed. As I have been told by Dr. Henry A. Barrett, New York, he has seen remarkable improvement of retinitis pigmentosa by the diathermic treatment. In combining the two methods we see a new possibility of helping in this serious disease.

Of many cases of retrobulbar optic neuritis which we have treated we have to report that a certain shortening of the course of the disease is possible if we introduce adrenaline from the nasal side into the globe. We take calcium chloride 1 : 300 with 3-5 drops of adrenaline (1 : 1,000) and put the tube far from the limbus into the orbit; dose 1-2 milliampères, 1·5-2 minutes every day. The optic nerve recovers from one sitting to another. In most of the cases after a week there was normal vision and operative interference like opening of the sinuses was unnecessary.

Résumé

The systematic study of the method of ionization reveals many interesting and new observations, of theoretical as well as practical therapeutic character. By introducing certain drugs, all being electrolytes, we are enabled to increase their pharmacodynamic effect and thereby obtain better therapeutic results. The method

of ionization is simple, its technique having been improved by new apparatus and new practical electrodes. Every practitioner in eye work may employ it with ease. We repeat once more: The method is of great therapeutic value in most of the common diseases and changes of the eyes and, when used in the uncommon and rare cases, sometimes produces astonishing and very impressive therapeutic results. If used in the early stage of a disease the restoration of the function is complete. But even in chronic cases the method should be tried since it stops the progress of inflammations and is a mighty factor in accelerating the resorptive power of the tissues.

LITERATURE

Arruga.—*Verein mitteldeutscher Augenärzte*, Halle, 1929.
Bos.—*Dissert.*, Groningen, 1929.
Erlanger.—*Zeitschr. f. Augenheilk.*, 1922; *Tierärztliche Rundschau*, 1926; *Klin. Wochenschr.*, 1928; International Congress of Ophthalmology, Amsterdam, 1929; *Jahreskurse für ärztliche Fortbildung*, 1932.
Erlanger, G., and Erlanger, Alice.—*Deutsch. med. Wochenschr.*, 1931; *Klin. Monatsbl. f. Augenheilk.*, 1932
Feldberg.—*Zeitschr. f. d. ges. Exp. Med.*, 1931.
Feldberg and Schilf.—Histamin. Berlin. Julius Springer, 1930.
Korrenjewitsch.—*Zeitschr. f. Augenheilk.*, 1929. Ref.
Nümi —*Klin. Monatsbl. f. Augenheilk.*, Ref., April, 1935.
Malkin.—*Klin. Monatsbl. f. Augenheilk.*, 1932.
Rochat.—International Congress of Ophthalmology, Amsterdam, 1929.
Schnyder.—*Klin. Monatsbl. f. Augenheilk.*, 1919.
Wirtz.—*Klin. Monatsbl. f. Augenheilk.*, 1908.
Cantonnet.—L'ionisation dans l'ophtalmologie. Paris, 1927.
Rousseau et Nyer —La practique de l'ionisation. Doin et Co., Paris, 1931.

SCLERECTOMY WITH IRIDENCLEISIS.—A MODIFIED GLAUCOMA OPERATION

BY

W. A. WILLE

JAVA

SEEING that there have been described so many good operations for glaucoma, I have been in doubt, whether I should publish the method which I have used during the last ten years. Still, as it has given good results, it might perhaps be of some use to describe it.

My idea has been to find a method that will combine perfect safety with great efficiency. It is in reality to a certain degree a combination of Herbert's wedge-isolation-operation and iridencleisis; but there are differences which prevent me from calling the operation after Herbert. These differences are the following:

1. I am not operating subconjunctivally, but exposing the operation-field by forming a conjunctival flap.

2. I combine the two operations mentioned above; and

3. I fasten the fold of the iris in order to prevent it from withdrawing into the anterior chamber, a mishap which otherwise often will take place. For this purpose I use a stitch. The only suitable material for this purpose is a woman's hair.* Further details are given in the description of the operation which follows.

First are given two instillations of pilocarpine and then 4 instillations of 5 per cent. cocaine, then pencilling of palpebrae and surroundings with tinct. jodii 1-spir. fort. 4. For injection I use a solution of cocaine and adrenaline (or supra-renine) of which 1 c.c. contains 2 milligrm. cocaine and 1 drop of a 1 per cent.

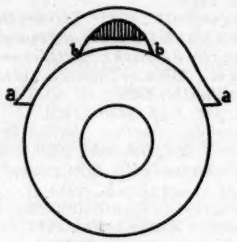


Fig 1.

solution of adrenaline. From this solution are given 2 c.c. retrobulbar, 1 c.c. subconjunctival on the upper part of the globe and 5 c.c. around the external canthus for akinesia. Then 4 instillations of cocaine and 2 of adrenaline.

The retrobulbar injection is very important; in the first place because it diminishes the tension considerably, and then for the anaesthesia and immobilization of the globe. After the injection we must wait 10-15 minutes before beginning the operation.

First. An external canthotomy is made. The conjunctiva bulbi is cut along the upper third part of the limbus. At both ends the incision is elongated 4 mm. in a horizontal direction. The flap is freely undermined and pushed away from the limbus (Fig. 1, a a).

Now, the wedge-isolation-operation of Herbert is made with the very narrow knife of the same author. The length of the flap is 7 mm. and the breadth 2 mm. (Fig. 1, b b). If the point of the knife should appear outside the limbus, it must be withdrawn and adjusted, otherwise the iris will easily get perforated.

* Women's sterile hairs are available in flacons (RM. 1.30) from R. Wurach, Berlin C2, Neue Promenade 5.

Before the incision has been quite finished, the top of the flap is cut off from behind.

This fragment, "the wedge," can be removed; but if it is a little adherent at a single point, it can also be left. It will not cause any difficulty. In Fig. 1, we see the flap b b after the top has been cut off.

For the following manipulation we must use Matthieux's iris forceps. With this instrument the iris is grasped half way between the root and the edge (Fig. 2). A fold of the iris is drawn out and perforated with a fine needle armed with a woman's hair

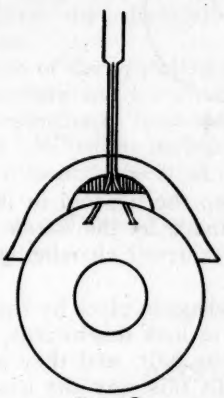


Fig 2

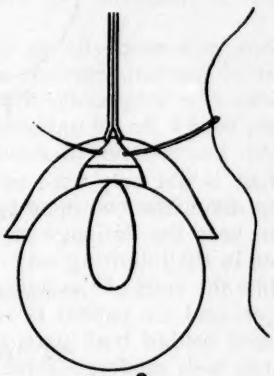


Fig 3

which is also provided with a needle at the other end (Fig. 3). Both ends are brought through the conjunctival flap from behind forward at a distance of about 7 mm. from the edge (Fig. 4). *The stitch need not be tied.* The ends are cut off in a length of 8 cm. and are fixed on the skin with leucoplast, but in no way stretched; on the contrary, they must be so long, that the eye can move freely in all directions without any stretching of the hair.

The conjunctival flap is drawn freely down over the cornea and fastened in this position by two stitches. After the operation has been completed, atropine must be instilled twice. This is important. It is advisable to close the canthotomy with a single stitch.

In Fig. 4, the conjunctiva bulbi has been drawn down over the whole operation field and fastened in this position by two stitches, c c.

The letters h h mark the free ends of the hair. The stippled lines show what is covered by the conjunctiva, viz., the sclerectomy-wound and the fold of iris, kept in position by the hair.

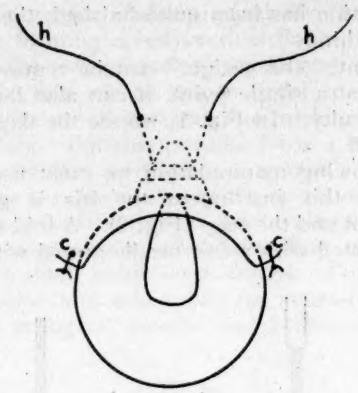


Fig 4

The hair is not only used to keep the iris-fold in its place, but also to dilate the two openings made by the needle and in this way to keep the drainage of the anterior chamber going. This is done in the following way:

While the ends of the hair are kept in place by the pressure of a finger, and the patient is told to look downwards, a glass rod is passed behind both parts of the hair, and they are stretched upwards with moderate force. In this way the iris-fold is also stretched upwards, and the holes in it are dilated. During one week this manipulation is repeated every day, and then the hair is removed.

In Fig. 5, we see a diagram of a sagittal section of the eye; a is the iris-fold, b one of the holes and c the conjunctiva bulbi.

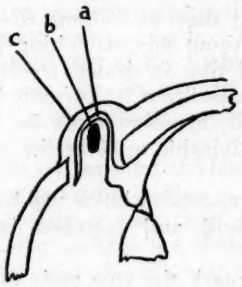


Fig 5

I imagine that somebody perhaps while reading this description would ask himself the question: "But what about infection?"

I fully understand this. In fact, I should never think of doing this operation when using a thread as sewing material—even if it were possible. All the fine voids in the thread would be hiding places for microbes and sources of infection. But a hair is a compact cylinder. And there is one thing more; the needle holes in the conjunctiva bulbi will very soon close firmly, "hermetically" around the hair.

However, more important than these considerations is the experience, and I am pleased to tell, that in 107 operations there has not been the slightest trace of infection or even of inflammation.

The results of the operation have usually been very good. In a few cases there was for a time a moderate increase of tension. In such circumstances treatment with pilocarpine will sometimes be sufficient. When the patient was living far off, I have preferred to perform a second operation opposite to the first and then with good result.

I can say, that time and experience have increased my confidence in the operation.

ABSTRACTS

I.—TRACHOMA

MacCallan, A. F. (London).—Trachomatous conjunctivitis, its surgery and pathology. *Lancet*, p. 215, January 25, 1936. Hunterian Lecture. R.C.S.Eng. on January 17, 1936.

MacCallan states that trachoma is a primary epithelial lesion probably due to a virus and that the histological changes seen in subepithelial aggregations, and sometimes diffuse exudations, of lymphocytes differ in no way from other forms of conjunctivitis such as follicular conjunctivitis. The characteristic features of trachoma are the extension of this subepithelial lymphocytic infiltration to the upper fornix and down to the upper corneal limbus, and the formation of pannus. Pannus is the result of direct spread and is not caused by contact with the infected upper palpebral conjunctiva.

The gelatinous swellings seen in stage 2 are not due to development and change in the original follicles but to occluded ducts of Meibomian and sebaceous glands with retention of their secretion.

Epithelial changes are at first proliferation then villous formation, ulceration and subsequently scar tissue epithelium. In some cases there are localised areas of papillary hypertrophy. Hyperaemia of the lid margin may lead to the development of supernumerary lashes and trichiasis.

Pannus begins as an indefinite grey infiltration at the limbus followed by tiny grey elevations 0·2 to 0·4 mm. in diameter. Capillary loops run centripetally from the vascular loops at the limbus. In severe cases Bowman's membrane is destroyed by the pannus and marked corneal opacities are formed.

The author defines the 4 stages into which he divides the clinical manifestations of trachoma. The aetiology and prophylaxis are discussed.

There are many helpful reflections and observations about the treatment of this disease. The author feels that the manipulative treatment of trachoma is best left to the expert, otherwise it is better to allow the patient to treat himself with drops and lotion than for him to submit to the attentions of a partly trained or inexperienced person.

For the gelatinous excrescences seen in stage 2 expression is recommended and for the 3rd stage the application of irritants such as copper.

Epilation of the lashes is condemned by the author, who also disapproves of the use of lid clamps and the removal of skin from the lids. He favours Streatfeild's operation in which a horizontal wedge-shaped strip is removed from the anterior surface of the tarsal plate and van Millingen's operation where a mucous membrane graft is inserted into the free border of the lid posterior to the lashes.

H. B. STALLARD.

II.—MISCELLANEOUS

(1) **Castan, J. M. H.—Syringomyelia and neuro-paralytic keratitis. (Siringomiela y Queratitis Neuroparalitica).** *Arch. de Oftal. Hisp.-Amer.*, Vol. IX, p. 419, 1934.

(1) **Castan** says that neuroparalytic keratitis commonly occurs after the removal of the Gasserian ganglion, or after alcohol injection of the fifth nerve for trigeminal neuralgia. A few days after either event there is a complete anaesthesia of the eye accompanied by corneal ulceration.

The aetiology is varied and interesting, and many theories have been formulated regarding the exact pathology of the condition. The most frequently recognised aetiology is a lesion of

the fifth nerve, and the cause may be traumatic or due to a tumour or meningitis.

40 per cent. to 50 per cent. of all cases of removal of the Gasserian ganglion suffer from this condition. Tabes, polyneuritis and syphilis are aetiological factors.

Gradertz described a case following poisoning by acetylene. Cases in which a sympathetic lesion is recognised are extraordinarily rare in the literature.

A participation of the sympathetic would revise the "trophic trauma" theory, which has been brought forward in support of a vasomotor lesion.

The author has found many cases of neuroparalytic keratitis where there have been lesions of both the sympathetic and the fifth nerve. A case has been recorded following removal of the larynx, and was attributed to the fact that ligature of the internal carotid had cut off the blood supply to the fifth nucleus, but surely the cervical sympathetic lesion was the cause? It has been shown that lesions of the fifth nucleus and the part of the nerve before it enters the Gasserian ganglion do not cause neuroparalytic keratitis.

The vasomotor theory is based on the presence of the sympathetic fibres which run with the fifth nerve.

Syringomyelia causes changes in the sympathetic nervous system, and in rare cases it has been known to cause neuroparalytic keratitis.

The author has met with Horner's syndrome in 25 per cent. of cases of syringomyelia, and more rarely heterochromia, hemiatrophy of the face, and one case of neuroparalytic keratitis.

Neuroparalytic keratitis starts with a superficial punctate keratitis. The cornea loses its brightness, and many small grey points appear, on which the epithelium appears to be heaped up and irregular, some of this disturbance is also in Bowman's membrane.

The condition spreads and exfoliation and loss of surface epithelium occurs in the centre of the cornea. Ulceration progresses until nearly the whole of the central area of the cornea is involved and a circular ulcer is produced. Occasionally the upper or lower segment is affected instead. Hypopyon may occur if the ulcer becomes infected and iritis accompanies it.

The disease is characterised by lack of any reactive symptoms, *i.e.*, pain, blepharospasm and lacrymation. If infection does occur the consequences may be serious.

The author's case was a man of 36 years, with syringomyelia, the left eye was normal, the right eye was enophthalmic and had ptosis. There was very slight conjunctival injection, nearly the whole surface area of the cornea was desquamated. There were

folds in Descemet's membrane and some changes in Bowman's membrane. The corneal reflex was much diminished, and the iris was hyperaemic. The pupil was small and active. There were no subjective symptoms and the patient had only come to hospital because his sight was bad.

The patient showed vaso-dilatation on his cheeks, ears and hands, the fingers of the right hand showed bruises, owing to loss of sensation. The syringomyelia was of the cervical type, C.S.F. and Wassermann test were negative. The author then had to determine whether there was any relation between the neuroparalytic keratitis and the syringomyelia.

In syringomyelia there is a proliferation of neuroglia accompanied by a vacuolation of the medulla. The symptoms are loss of sensibility to heat, cold and pain, with preservation of sensation of touch, at the same time there are trophic and vasomotor lesions.

The cervical sympathetic, the trigeminal and the vestibular nuclei and the nuclei of the ocular nerves may be affected. There may be nystagmus due to lesions in the vestibular nucleus. The sixth nucleus is most frequently affected.

Changes in the pupil reactions are common; the other eye symptoms have been mentioned previously. In the author's case the pupil was contracted, the palpebral aperture diminished and the eye enophthalmic, there was marked ptosis, the facial vessels injected, the skin temperature raised and excessive sweats occurred.

There was no fall of ocular tension, as sometimes occurs. Which was responsible in this case, the trigeminal lesion or the sympathetic ?

Against the sympathetic being responsible is the fact that sections of the sympathetic and removal of the cervical gland do not cause neuroparalytic keratitis, but many lesions of the trigeminal and Gasserian ganglion are not followed by this condition. For example there are cases of complete anaesthesia of the cornea (due to trigeminal paresis) which remain *in statu quo* for years and never develop neuroparalytic keratitis. A case has been described of a fractured base with complete fifth nerve paresis and lagophthalmos with facial paresis and no neuroparalytic keratitis. A case of trigeminal paresis with trichiasis never developed neuroparalytic keratitis.

Occasionally the cornea is hypersensitive in neuroparalytic keratitis, in fact neuroparalytic keratitis has occurred with an anaesthetic or a sensitive cornea, with a dry cornea or with good lacrymal secretion, with ptosis or with lid retraction. Neuroparalytic keratitis does not occur with fifth nuclear lesions or lesions of the nerve trunk.

So other authors have sought for an explanation in vasomotor and trophic changes or anything interfering with the nutrition of the cornea.

Vasomotor and trophic changes are observed in most cases of neuroparalytic keratitis, and sympathetic nerve fibres run with the fifth nerve. A paralysis of the sympathetic has been observed after removal of the Gasserian ganglion. The author thinks that the sympathetic fibres that run with the fifth nerve are responsible for most cases of neuroparalytic keratitis attributed to the fifth nerve.

Behr says that the keratitis is produced by an abnormal trophic reaction, and is due to an affection of the ganglion cells.

The author concludes in his case that the syringomyelia caused the sympathetic changes and these gave rise to considerable vasomotor and trophic disturbances in the eyes, hands and ears.

Because neuroparalytic keratitis has appeared in a cornea protected by ptosis and with much sympathetic paresis, in the author's "humble opinion" the aetiology in his case is the trophic vasomotor changes, produced by the sympathetic lesion during the course of a syringomyelia of the cervical type.

E. E. CASS.

(2) **Young, C. (Roanoke).—Friedreich's ataxia showing transitional symptoms of Marie's disease (familial spinocerebellar ataxia).** *Trans. Amer. Ophthal. Soc.*, Vol. XXXII, p. 626, 1934.

(2) **Young** describes 2 cases of ataxia affecting 2 young males in the same family and in whom were present clinical features of Friedreich's ataxia and Marie's disease (familial spinocerebellar ataxia). These cases were essentially of the Friedreich type but showed some transitional signs of Marie's disease.

Nystagmus, a constant feature of Friedreich's ataxia but absent in Marie's disease, was present to some extent in both patients and optic atrophy, which is an early and quite constant sign in Marie's disease but absent in Friedreich's ataxia, was also evident.

In one case exophthalmos of 1mm. was noted in the right eye and the author comments on the fact that this physical sign has hitherto not been recorded in the literature of this disease.

The neurological features of ataxia, standing on a wide base, the gait, pes cavus, dorsal extension of the big toes, and altered tendon reflexes are recorded.

The literature, aetiology and points in differential diagnosis of Friedreich's ataxia and Marie's disease are discussed.

H. B. STALLARD.

(3) **Lloyd, R. (Brooklyn).—Arachnodactyly. (Dystrophia mesodermalis congenita, typus Marfans. Marfan's syndrome. Dolichostenomelie).** *Trans. Amer. Ophthal. Soc.*, Vol. XXXII, p. 361, 1934.

(3) **Lloyd** in this paper reviews the main clinical features of arachnodactyly and discusses the ocular disorders associated with this defect. Congenital miosis and bilateral ectopia lentis are chief among these. In the former the pupil will not dilate with atropine and the latter complication was noted in 5 or 6 cases seen by the author and according to the literature occurred in half the number of cases of this disease. Two Italian families are described by the author, 5 out of 8 persons were affected in one family and 4 out of 6 in the other. In one family the defect was traced to the mother and the maternal grandfather.

The lens is generally displaced up and out and in some cases has dot-like opacities. The fibres of the suspensory ligament are clear and strong, and fine red-brown dots were seen on these and in the vitreous.

In 2 cases quoted from the literature the lens was removed after dislocation into the anterior chamber. It was found to have the weight and diameter of the lens in a new-born infant, increased thickness, and defective development at a period in later intra-uterine life. On the posterior capsule there were remains of embryonic tissue and a fissure at the pole in one case. The fibres of the lens were arranged irregularly. In another lens there was heaping up of the nuclei in the posterior part of the lens and pathological changes suggestive of an over-development of the anterior portion of the lens.

In the discussion that followed this paper Vail made the observation that arachnodactyly was sometimes associated with megalocornea, a deep anterior chamber, persistent pupillary membrane, atrophy of the superficial layers of the iris and was a progressive disease.

H. B. STALLARD.

(4) **Swab, C. (Omaha).—Tuberculous and Streptococcic retinal haemorrhages.** *Trans. Amer. Ophthal. Soc.*, Vol. XXXII, p. 388, 1934.

(4) **Swab** discusses the aetiology of retinal haemorrhages and champions the cause of tuberculosis as an aetiological factor in the production of perivasculitis of the retinal vessels and subsequent haemorrhages. He reports 5 cases, the ages of the patients varied from 22 years to 46 years, 3 were females and 2 males. In the patient aged 46 years the vascular changes resembled those seen in hyperpiesis. This case and 2 others

responded to tuberculin therapy and apparently one case required this treatment to prevent recurrences. Two other cases cleared up after the removal of focal sepsis and administration of autogenous streptococcal vaccine. All the tuberculous cases showed retinitis proliferans whereas the vitreous became clear in one of the streptococcal cases and manifested a " dense, grey fibrous sheath" in the other. In the 4 younger patients the retinal veins were involved and in the older (aged 46 years) the arteries were affected more than the veins.

The author claims that the 'clinical experience' of response to tuberculin therapy justifies the diagnosis of a tuberculous cause for the retinal and vitreous haemorrhages.

H. B. STALLARD.

(5) **King, Clarence (Cincinnati).—A case of chloroma with orbital involvement locally benefited by X-ray therapy.** *Trans. Amer. Ophthal. Soc.*, Vol. XXXII, p. 340, 1934.

(5) **King** has described a case of chloroma in the orbit of a boy aged 9 years who had left exophthalmos, enlarged cervical lymph nodes, an enlarged liver, a palpable spleen and swellings in the scalp and alveolar margins. He was anaemic and had a sallow complexion.

The swelling in the left orbit disappeared in 25 days after X-ray therapy. Papilloedema and haemorrhages around the macular region and in the inner layers of the retina were present in both eyes. Ulcerative pharyngitis, tonsillitis and bronchopneumonia terminated in death.

The author comments on the rarity of chloroma lesions affecting the eyes and orbit. Post-mortem examination revealed much fibrosis and cellular degeneration in the orbital tissues on the affected side. The other lesions in the scalp and elsewhere were composed of myelogenous cells in a very sparse connective tissue stroma. The author quotes Cunningham's views about the importance of supra-vital staining methods in examining affected tissues and the blood in order to avoid mistaking young abnormal myelocytes for small lymphocytes.

A discussion on the pathology of chloroma is included in this paper. The author quotes Lehndorff's view that chloroma belongs to a group of borderline cases between genuine neoplasms and leukaemias and that Sternberg maintains that chloroma is a leucosarcoma or myelosarcoma. He mentions recent experimental work by Fischer-Wasels and Bungeler who have produced leukaemia and lymphosarcoma experimentally by long continued administration of indol. They believe that the myeloses and lymphadenoses represent real neoplasms of the blood forming tissues.

H. B. STALLARD.

(6) **Giannini (Pisa).—"Punctate albino fundus" and its relation to Oguchi's disease. (Contributo clinico allo studio del "fondo albino punctato" e considerazioni sui rapporto colla malattia di Oguchi).** *Ann. di Ottal.*, September, 1934.

(6) Oguchi's disease is a rare familial condition, seldom met with except in the Japanese; the chief symptom is night-blindness which does not alter with years; and the retinal condition is a diffuse whitish grey coloration of the fundus, allowing the vessels to stand out conspicuously, apparently darker than usual by contrast. Mizuo noted that after prolonged dark adaptation, the retina regained its normal appearance and the light sense improved temporarily. Giannini describes two cases of "punctate albino fundus" and separates them from retinitis punctata albescens since they have no contraction of the visual field and no disturbance of colour vision. As to the pathology of the disease, nothing is certain. It seems probable that both the discoloration and the loss of function of the retina depend on some disturbance of the photochemical change which has been demonstrated to occur in the outer segments of the rods; it may be that exposure to light develops an opaque substance in the rods, which limits their activity and accounts for the change of colour of the fundus: when in darkness this substance disappears, the fundus resumes its colour and the retina its function.

HAROLD GRIMSDALE.

(7) **Accardi (Modena).—The so-called blockage of the reticulo-endothelial system and its effect on the intra-ocular tension and the condition of the aqueous (Ulteriori ricerche sugli effetti del cosidetto blocco del S.R.E. sulla tensione endoculare, tasso albuminoideo ed indice di rifrazione dell'umore acqueo).** *Rass. Ital. d'Ottal.*, July-August, 1934.

(7) To produce the block **Accardi** chooses a substance whose toxic action on the tissues is practically nil; colloid sulphide of mercury. This substance if introduced in colloid form rapidly changes to solid granules and remains fixed in the tissues, undergoing only very slow change to oxide. With these injections, the author found that the intra-ocular tension constantly rose, and the refractive index and albuminoid content of the aqueous, rose also. To prove that it was the block that gave rise to these signs, he injected other animals with equivalent quantities of gelatine, and others with a solution of sublimate of mercury; these animals showed no rise. These experiments confirm the important relationship between conditions of the vascular system and the intra-ocular tension.

HAROLD GRIMSDALE.

(8) **Padovani (Naples).—Endocrinal irregularity in a patient suffering from spring catarrh and associated keratoconus. (Aritmia endocrina in soggetto primaverile con cheratocono).** *Lett. Oftal.*, September, 1934.

(8) The relative frequency with which spring catarrh is associated with conical cornea suggests some definite causal relation between the two diseases. Padovani's patient was a man of 22 years who had suffered from spring catarrh since the age of 16 years. He was seen in November, 1933, saying that the acuity of his right eye had failed: R.V. 1/150. The left eye then showed no corneal change but in January a definite conical ectasia was observed in the left eye and vision had fallen to 1/6. An examination gave signs of endocrinal insufficiency, particularly of the pituitary, thyroid and suprarenals.

HAROLD GRIMSDALE.

(9) **Tristaino (Catania).—Investigation of the reticulo-endothelial system by means of Congo Red in trachoma. (Esplorazione, col metodo del rosso Congo, del sistema reticulo-endoteliale nei tracomatosi).** *Boll. d'Ocul.*, October, 1935.

(9) It has been shown that this system of tissues has very important functions in protecting the body against infection.

Its various parts are more active in the face of infective and parasitic disease. It has also been shown that the cells of the reticulo-endothelial system take up Congo Red when this is injected into the circulation, in quantity varying directly with their activity; thus in the presence of disease, when the cells are specially active, the Congo Red disappears from the blood more rapidly and completely than in normal health. It has been held by many, and especially by Angelucci and his school, that trachoma is in some sense the local expression of a general lymphatic hyperplasia; and that it is in some way connected with tuberculosis.

Tristaino has experimented in a number of cases of trachoma in various stages, injecting Congo Red and examining the blood after the lapse of an hour, to find how much has been taken up by the tissues. He finds an almost constant high level of ingestion, showing an activity of the reticulo-endothelial system above normal, and therefore suggesting some general morbid state; the reaction is very similar to that found in tuberculous patients and he thinks that this serves to make more probable a definite relation between the two conditions.

HAROLD GRIMSDALE.

(10) **Scullica (Messina).—Avitaminosis and xerophthalmia. (Avitaminosi e sindrome xeroftalmica).** *Ann. di Ottal.*, November, 1935.

(10) It is not very rare to observe small series of cases of xerophthalmia but whereas these are usually seen in spring, **Scullica** noted a small epidemic in the autumn of 1931. It seems clear that these cases are often due to a diet deficient in vitamin A; this is provided normally in the food which contains some substance or substances which are converted by the liver into vitamin A. When the liver is put out of action, by disease or experimentally, the syndrome due to avitaminosis is often seen.

The conjunctival lesions seem to be due partly to trophic changes in the membrane and partly to infection by bacteria. The night blindness depends on the delay of regeneration of retinal purple. It has been supposed that the visual purple takes origin from vitamin A; this is a pure hypothesis, though the vitamin has been shown to be a normal finding in the retina. The author saw in the majority of his cases changes in the pigmentation of the retina. There were in many small whitish areas where the pigment seemed lacking; this change lasted after the night blindness had been relieved.

HAROLD GRIMSDALE.

(11) **van Poole (Honolulu).—Leprosy and tuberculosis of the eye.** *Trans. Amer. Ophthal. Soc.*, Vol. XXXII, p. 596, 1934.

(11) **van Poole** in describing the histology of leprous lesions draws attention to certain pathological features similar to those seen in tuberculosis such as the intercellular and intracellular presence of the acid-fast bacilli, the extension of the disease by the lymph and blood streams and the appearance of certain chronic inflammatory cells common to these chronic granulomata.

The author gives a brief account of the large polygonal cells which contain vacuoles, one or more poorly staining nuclei and brownish coloured clumps of bacilli. Sometimes these cells are arranged in an alveolar pattern with collagenous centres, sometimes in broad columns around the papillae, sweat glands, hair follicles and nerves.

The paper describes leprous lesions of the lids, eyebrows, nerves, orbicularis muscle, sclera, cornea, anterior chamber, iris, ciliary body and the anterior part of the choroid and retina.

The author comments on the fact that leprosy like tuberculosis affects mainly the anterior part of the eye. He discusses the endogenous route of ocular infection.

In 206 cases of leprosy he has observed hyperaemia, venous congestion and blurring of the optic disc margins, particularly

on the nasal side, in 49 cases (23·7 per cent.). He quotes Santonastaso who has seen such changes in 26 out of 50 cases. The author believes that these changes in the optic disc which he refers to as optic neuritis are possibly caused by an allergic reaction to the protein of the Hansen bacillus. Many of the patients in whom he made this observation also manifested signs of a general allergy. He quotes Bieling and Oelrichs who have stated that members of the acid-fast group of myco-bacteria are alone capable of inducing a generalised allergic state in which all the body tissues are involved.

Thirty-one out of 49 of the author's cases showing optic disc changes were uncomplicated but the remaining 18 had nephritis, diabetes or lues.

At the end of this paper there is a short account of 260 patients from mixed races who exhibited tuberculous lesions. In 3·4 per cent. of these were the fundus lesions solely due to tuberculosis.

H. B. STALLARD.

(12) **De Sanctis (Parma).—The initial symptoms of interstitial keratitis in hereditary syphilis. (Sulla sintomatologia iniziale della cheratite parenchimatosa eredoluetica).** *Boll. d'Ocul.*, September, 1933.

(12) It is generally agreed that in all cases interstitial keratitis affects not only the cornea but that the uvea takes part in the inflammatory process also. **De Sanctis** has had the opportunity of observing the onset of the disease in a number of patients in whom the one eye was already attacked and the second became inflamed while under observation. He examined the unaffected eye from day to day, while the patient was attending the clinic, by means of the slit-lamp, and finds that in all cases the disease begins as an inflammatory process of the anterior part of the uveal tract.

HAROLD GRIMSDALE.

(13) **Zavalia, A. U. and Oliva, R. O. (Cordoba).—A case of primary carcinoma of the lacrymal sac. (Sobre un caso de carcinoma primitivo del saco lagrimal).** *Arch. de Oftal. de Buenos Aires*, Vol. X, p. 521, 1935.

(13) **Zavalia** and **Oliva** say that primary malignant tumours of the lacrymal sac are very rare; cases of lymphoma, sarcoma, plasmocytoma and epithelioma have been described, but in the literature in all only 50 cases have been recorded.

They describe two cases that they themslves know of.

The first was a man aged 36 years, who for a year had suffered from epiphora in the left eye; he had no other symptoms; and did not pay any attention to this condition until a tumour

began to develop in the region of the lacrymal sac. On examination this tumour was found to be ovoid in shape, was quite painless and felt as though it was a collection of tense fluid; it was not adherent to the skin, which appeared to be quite normal. On pressure, however, there was no regurgitation through the lacrymal passage or through the nose.

A mucocele was diagnosed but at operation it proved to be a solid tumour of the sac which was removed without any difficulty, and sent for section. It was described as a malignant tumour of epithelial origin.

On account of the nature of the tumour an application of X-rays was given; but the patient then disappeared as he felt perfectly well, and was not seen again until 3 months later. There was then another tumour about the same size as that which had previously been removed and this was treated by radium. The patient left the country and was treated by Dr. Sena at Buenos Aires, and after having radiotherapy was considered to be cured, but 8 months later extensive cicatrisation involving both lids, the left side of the nose and a large part of the cheek, with total symblepharon and atrophy of the eye occurred. Recently the authors heard of the death of this patient, but, unfortunately, do not know the cause.

The second was a man aged 47 years who had suffered from a painless epiphora without any other signs. For a year he had noticed a small, painless, hard tumour in the region of the lacrymal sac.

On examination this tumour was found to be the size of a small nut; it was sessile and was pushing the lower lid up so that the palpebral aperture was diminished; it was painless and on examination was found to have invaded the orbit, in the region of the lower-nasal angle.

The skin which covered the tumour was a little erythematous, but not adherent, the palpebral conjunctiva was reddened and hypertrophied; the lacrymal passages, however, in this case were freely open. There was one hard enlarged gland in the angle of the jaw.

A small portion of the tumour was excised for microscopy and was reported to be carcinomatous. The tumour was removed surgically, and some days later radium was applied. For 6 months the patient appeared to be cured, but he then developed a small tumour in the left lower lid. This tumour was removed.

It was very adherent to the periosteum, and was extending into the orbit. After some days radium was again applied. Up to the present, i.e., 4 months later, there has been no recurrence.

The authors stress the importance of diagnosing the condition as early as possible.

Until recently the only treatment employed for these tumours was surgical, but there is no doubt that the application of high frequency currents in the modern methods of electro-surgery will make it more probable that the carcinomatous cells will be destroyed and diminish the danger of a recurrence.

But whether they are operated on or not, the authors consider that they should always be treated with X-rays or preferably radium.

E. E. CASS.

(14) **Baurmann, M. (Göttingen).—Degenerative processes in the vitreous of the human eye and their relation to the origin of retinal tears. (Zerfallsvorgänge im Glaskörper des menschlichen Auges und deren Beziehung zur Entstehung von Netzhautrissen).** *Arch. f. Ophthal.*, Vol. CXXXIV, p. 201.

(14) **Baurmann** here puts forward a view of the causation of retinal tears in the region of the ora serrata based on a study of the vitreous of four clinically normal eyes of patients between 16 and 55 years of age with the ultra-microscope; in all these he found definite degenerative changes in the form of cavities or spaces filled with fluid, and granular material between the adjacent fibrils which in places were markedly coarser in texture, these changes increasing in extent with the age of the patient.

It is this tendency to degeneration of the vitreous and its firm attachment to the retina at the ora serrata and orbiculus ciliaris that accounts for tears at the anterior limit of the retina, due to the traction that may be exerted by the vitreous on the retina in this region on movements of the eye.

Where the vitreous is either completely liquified or is a uniform gel there would be no pull on the retina on movement of the eye, but in cases of advanced liquefication of the vitreous, where only small portions of normal structure remain attached to the retina in front, the greater, liquefied, part of the vitreous remains stationary on movements of the eyeball, but the normal parts move with it and pull on the retina at the point of attachment, thereby causing a tear, with a force that is calculated according to the area of vitreous attachment and the velocity of the ocular movements.

THOS. SNOWBALL.

(15) **Fontana (Modena). — Glycogen in the retina. (Ulteriori ricerche istologiche sul glicogeno della retina di alcuni vertebrati allo stato normale de in talune condizioni sperimentali).** *Rass. Ital. d'Ottal.*, March-April, 1935.

(15) **Fontana** has repeated his researches on the position of glycogen in the retina, making use of the methods of Best and Langhans instead of that of Fischer with which his previous

experiments were made. The present confirm his former findings.

In the rabbit and in the frog, glycogen is found in the pigmented epithelium, in the rods and cones and in the external limiting membrane. No change in the amount of glycogen is found under different conditions of light except in the pigment epithelium; here it is increased largely after exposure to strong light.

The author produced experimental hypo- and hyperglycaemia (the one by injections of insulin, the other by injection of pilocarpine) and found glycogen diffused through all the layers of the retina in each case.

HAROLD GRIMSDALE.

(16) **Colomba (Padua).—Colour vision in various affections of the optic nerve. (Comportamento del senso cromatico nelle varie affezioni del nervo ottico).** *Boll. d'Ocul.*, April, 1935.

(16) Examination of the macular colour sense by means of the chromatic equation, has sometimes shown a reduction of perception of green in cases where there is known to be affection of the optic nerve, though the form vision is normal, and the ophthalmoscope shows no fundus change. But little attention has been paid to variations of the peripheral colour sense in disease. Lo Cascio has devised a chromatoscope by which can be estimated the amount of coloured light which must be added to white light of constant intensity to produce the sensation of colour. **Colomba** has used this apparatus in the investigation of the colour sense of a number of patients having definite disease of the optic nerve, and publishes tables to show the degree of defect. His conclusions are as follow; (1) colour sense is reduced, both in the macular and the peripheral zones to some extent, in all cases of disease of the optic nerve. (2) In tabetic and descending atrophy, green is lost more than any other colour. (3) There is no close relation between central acuity and colour sense. (4) The visual field usually has a condition corresponding to the state of the colour sense, but sometimes colour sense is reduced though the field for white is normal.

HAROLD GRIMSDALE.

(17) **Anastasi (Florence).—The anaesthetic power of cocaine in relation to the pH. of the solution. (Il poter anestetico del cloridrato di cocaina in rapporto alla concentrazione idrogenionica della soluzione).** *Boll. d'Ocul.*, November, 1935.

(17) Regnier in 1924 announced that he had been able to increase the anaesthetic power of cocaine by administering it in alkaline solution. In 1933 Baldoni made similar experiments

with novocaine and percaine and announced similar results; all these experiments seem to have been made on animals. **Anastasi** has repeated the experiments using very dilute solutions of cocaine (0·25 per cent. and 0·5 per cent.) adding a varying quantity of alkali, and has tested their anaesthetizing power on the human cornea by noting the reduction of the lid-reflex on irritation of the cornea. He comes to the conclusion that with the solutions employed, the greatest anaesthetic effect was produced by the simple solution and that as the pH increases with the addition of alkali, the effect diminishes. It is known that any salt, to have any action on the organism, must undergo ionic dissociation and it seems probable that the presence of the alkali impedes this progressively with the increase of pH.

HAROLD GRIMSDALE.

(18) **Fontana (Modena)—The action of carbolic acid on the eye. (L'azione del fenolo sull'occhio).** *Rass. Ital. d'Ottal.*, July-August, 1935.

(18) The reason for the researches quoted in this paper was a statement that " Lacmin " (Ruata's antitrachomatous vaccine) was dangerous to the eye on account of the carbolic acid which it contained. **Fontana** has tested the action of phenol in solution of strengths from 0·25 per cent. to 6 per cent. dropped into the eyes several times daily for a fortnight. He finds that solutions up to 2 per cent. are incapable of producing any reaction; that from 2·5 per cent. to 3 per cent. solutions produce a slight hyperaemia which disappears in a few hours. In rabbits stronger solutions produce intense hyperaemia with much secretion, and some corneal desquamation. Since " lacmin " contains only a very small percentage of phenol the author concludes that the alterations sometimes observed after its use, cannot be due to the phenol.

The weaker solutions were tested on healthy and trachomatous eyes.

HAROLD GRIMSDALE.

(19) **Goulden, C. (London).—The principles of orthoptic training.** *Trans. Ophthal. Soc. U.K.*, Vol. LV, p. 576, 1935.

(19) **Goulden** in the early part of this lecture quotes liberally from the works of Buffon (1743) in the " Mémoires de l'Academie des Sciences " and from his book " Histoire Naturelle de l'Homme," passages of which are rich with sound observations about all the essential features of strabismus. The author's lecture contains a well-balanced survey of present views concerning strabismus especially its physiological aspects.

He describes the general principles for piloting a case of

strabismus from amblyopia, suppression, abnormal retinal correspondence and false projection to restoration of vision and the achievement of binocular vision. Technical details and descriptions of the large number of instruments employed in orthoptic training are omitted.

In discussing the indications for surgery and the correct time at which these measures should be undertaken he says "I think it should be remembered that concomitant squint is the derangement of the perception of convergence, and has not its primary cause in the muscles of the eyes. Operations upon the muscles of the eye would seem to be tackling the problem from the wrong end, and they are, after all relatively gross measures to try to correct such a very delicate mechanism as the correlated movements of the two eyes. We are not yet in the position to make a considered judgment as to the final results of orthoptic training combined with operation.

There will be a need to follow these cases for several years, and the opportunity for this has not yet come."

H. B. STALLARD.

(20) **Wolff, E. (London).—The causation of amblyopia following gastric and other haemorrhages.** *Trans. Ophthal. Soc. U.K.*, Vol. LV, p. 342, 1935.

(20) **Wolff** reviews the literature on this subject and comments on the clinical facts of these cases in the majority of which amblyopia occurs after repeated haemorrhages, particularly from such organs as the gastro-intestinal tract and the uterus. Cases have been recorded where blindness followed repeated venesection, after a single haemorrhage and in 2 cases during the Great War where it occurred following a secondary haemorrhage.

The author quotes the pathological investigations of Ziegler, Görlitz and Seese who found degenerative changes in the ganglion cells, cytoid bodies in the nerve fibre layer, retinal oedema and fatty degeneration in the optic nerve fibres confined to a region behind the lamina cribrosa (Görlitz's case) 22, 11 and 5 days respectively after the onset of blindness.

The author suggests that the changes leading to blindness are the result of defective oxygenation. In support of this he contends that the haemoglobin content of the blood is lowered in cases of gastric ulcer and uterine haemorrhage and that a further reduction of the haemoglobin leads to spasm in the central retinal artery, damage to the endothelium of the retinal capillaries and subsequent retinal oedema and degeneration of the ganglion cells and nerve fibres.

He quotes the experimental work of Uhthoff who clamped the optic nerve previous to excision of the eye and noted contraction

of the central retinal artery some 5—10 seconds afterwards. This fact suggested that the arterial contraction or spasm was not produced by the cutting off of the blood supply directly but by the fall of the oxygen supply below a certain point.

H. B. STALLARD.

(21) **Mann, Ida C. (London).—A theory of the embryology of oxycephaly.** *Trans. Ophthal. Soc. U.K.*, Vol. LV, p. 279, 1935.

(21) **Miss Mann** has brought forward an embryological theory in explanation of the changes seen in the eyes and skull in oxycephaly. The visceral mesoderm plays an increasingly important part in the development of the skull on ascending the animal scale to mammals and man. Its texture is denser than the paraxial mesoderm. The alisphenoid, pterygoid and temporal bones are developed from visceral mesoderm called into being by the lateral growth of the brain and accompanied by narrowing of the angle between the optic nerves. She states that oxycephaly is a dysgenic condition showing itself as a localised arrest of development of the extreme anterior portion of the visceral mesoderm (maxillary process) possibly of atavistic significance. Since the eyelids, eyes and other orbital contents (many of which arise in paraxial mesoderm) appear much earlier in phylogeny than the lateral expansion of the brain we should not expect them to be abnormal.

The optic atrophy which is evident in some cases of oxycephaly is probably the result of kinking of the optic nerves on their steep passage up from the abnormally depressed middle fossa to the intra-cranial openings of the optic canals, or the result of papilloedema from raised intra-cranial pressure early in life.

The authoress's theory supports such clinical features of oxycephaly as the wide distance the eyes are set apart, shallow orbits, exophthalmos, the prognathous jaw and high palate.

H. B. STALLARD.

(22) **Sorsby, A. (London).—Ocular lesions in bony dystrophies.** *Trans. Ophthal. Soc. U.K.*, Vol. LV., p. 499, 1935.

(22) **Sorsby** classifies certain cranial deformities subdividing those generally included under the term oxycephaly. He discusses ocular defects and orbital abnormalities in association with these anomalies.

Exophthalmos and ophthalmoplegia are noted in cases of acrocephalo-syndactyly. In dysostosis cranio-facialis where there is a boss in the line of the frontal suture, a parrot beak nose, sunken atrophic upper jaw and prognathous lower jaw, exophthalmos and divergent strabismus have been observed as in oxycephaly.

The orbits are situated laterally in cases of hypertelorism, a cranio-facial deformity, the result of early ossification of the lesser wings of the sphenoids. Divergent strabismus, mental deficiency, syndactyly, undescended testis, high arched palate, enlarged terminal phalanges and congenital cardiac disease are other defects seen in association with this anomaly.

The author describes an unnamed syndrome consisting of a large skull, ugly face, coarse hair, mental deficiency, corneal opacities in the anterior part of the substantia propria and enlargement of the spleen and liver.

One case of unilateral exophthalmos associated with cranio-cleido-dysostosis has been recorded in the literature, and brachydactyly and micro-corneae have been noted in a father and son.

The ocular defects seen in association with arachnodactyly, polydactyly and retinitis pigmentosa, the Laurence-Moon-Biedl syndrome, syndactyly with aniridia, apical dystrophy of the hands and feet and macular coloboma, and fragilitas ossium and blue sclerotics are also mentioned in this paper.

H. B. STALLARD.

(23) **Hohn, E. (Copenhagen).—Iridocyclitis and ribbon-like keratitis in cases of infantile polyarthritis. (Still's disease).** *Trans. Ophthal. Soc. U.K.*, Vol. LV, p. 478, 1935.

(23) **Hohn** reports the case of a female child aged 5 years with iridocyclitis and ribbon-like keratitis complicating Still's disease. The interphalangeal joints, wrists, knees and ankles were swollen, painless and showed impaired motility but no osseous change. The author quotes only one other case recorded in the literature.

He discusses the ocular complications accompanying other forms of chronic polyarthritis in childhood due to sepsis and 'rheumatism.'

H. B. STALLARD.

(24) **Behr, Carl (Hamburg).—A contribution to the study of the anatomy of, and clinical problems relating to, the septal tissue and the vascular system in the optic nerve. (Beitrag zur Anatomie und Klinik des septalen Gewebes und des Arterieneinbaus im Sehnervenstamm).** *Arch. f. Ophthal.*, Vol. CXXXIII, p. 227.

(24) This research by **Behr** is intended to supply a fuller knowledge of the finer anatomy of the optic nerve and its blood supply, and thereby throw more light on various physiological and clinical problems.

In summing up his findings the author refers to the fact that the ophthalmic artery is not the only source of blood supply to the orbit and optic nerve; between the lacrimal and the middle meningeal arteries there is an anastomosis through the sphenoidal fissure which may be the main artery to these parts.

All vessels supplying the optic nerve penetrate into it from the pial sheath. From the moment that the arteria centralis retinae passes through the pial sheath into the nerve it gives off no branches for its blood supply; it is strictly a retinal artery. The clinical picture of embolism of this artery may, therefore, arise by a block in the vessel within the nerve far behind the retina.

The septal framework of the nerve serves a double function, mechanical (being strengthened specially where protection of the nerve is most required through movements of the eye) and nutritive, in that it is the sole carrier of the vessels supplying the nerve fibre bundles. It is most dense behind the globe and just before and in the optic canal, less dense in the central intra-orbital portion and just behind the canal, while in the posterior intracranial part the septa close to the chiasma disappear.

While in these various parts of the nerve the arrangement and density of the septa is very uniform, there is in the intracranial portion a marked change in the septa running axially in which the papillo-macular bundle runs. They are thinner and less abundant than those in other parts, so that the nerve fibre bundles are there much thicker than elsewhere in the nerve. The finer septa for carrying the capillaries being also nearly absent here, the conditions for the nutrition of this bundle in this portion of the nerve are not so favourable as in other parts. This anatomical peculiarity offers a satisfactory explanation of the origin of retrobulbar axial optic neuritis or the central scotoma in toxic amblyopia, multiple sclerosis and the Foster-Kennedy syndrome.

The blood supply to the optic nerve is derived from three systems of vessels which freely anastomose with one another, (1) Branches from the posterior ciliary arteries around the lamina cribrosa: (2) The anterior arteries of the optic nerve, which penetrate the nerve with the central artery for the anterior and middle thirds of the intra-orbital portion: (3) The posterior arteries, branches of the ophthalmic artery, given off at the apex of the orbit, for the posterior third.

The central vessels in their passage through the dural sheath are not bound fast to the dura by their adventitia, but are surrounded by a system of fine spaces or channels (quite independent of the perivascular lymph system) which permit of a flow of cerebral fluid from the subdural into the epidural spaces. It is, therefore, impossible for the cerebral fluid to produce such a rise of pressure in the subarachnoid space in increased intracranial pressure as to prevent a return of venous blood from the retina and papilla by compression of the central vein, or to allow of its being pressed alongside the central vessels into the papilla.

All vessels passing from the orbit into the nerve (including the central vessels) are enveloped in a connective tissue covering as

they pass from the dura to the pia; this mantle, derived from the dural sheath, is bound more or less closely to the adventitial coat, and shuts off the vessels and their perivascular spaces from the subarachnoid space and liquor cerebralis; it merges into the pial sheath as the latter accompanies the vessels into the nerve. The central vessels lie in a groove on the inner surface of the dural sheath in their short sagittal course in the subdural space, an arrangement which prevents any pressure on the nerve by the pulsations of the artery and the compression of the vessels by the nerve.

During its course from its point of exit from the ophthalmic artery to its distribution in the retina the central artery takes at five places a change in direction which may amount to 90°.

THOMAS SNOWBALL.

(25) **Tisconia, A. and Tisconia, B. J. (Buenos Aires).—Congenital familial anophthalmos. (Anoftalmos congenito familiar).** *Arch. de Oftal. de Buenos Aires*, Vol. X, p. 355, 1935.

(25) The two **Tisconias** say that few cases of congenital familial anophthalmos have been recorded.

In anophthalmos there is a complete failure of development of the eye which is usually bilateral, although in some cases there is unilateral anophthalmos with microphthalmos of the other side. The cause is due either to an arrest or upset of development of the optic vesicles. Some authorities say it is due to destruction of the vesicle, whereas others say that the vesicle is never formed.

Various degrees of microphthalmia and anophthalmia have been produced experimentally. The cause of the condition in man is thought to be chronic infection such as tuberculosis or syphilis.

In a family of nine children who were examined by the authors, four had congenital anophthalmos; the parents were healthy and denied venereal disease, but unfortunately they would not submit to a bacteriological examination of the blood.

All cases showed the typically small palpebral aperture, and all had complete absence of both eyes; in two out of the four cases in one orbital cavity was found a very small lump the size of a grain of maize, which the authors consider was the rudimentary remains of the eyes.

In all other respects the children were normal and showed no signs of congenital syphilis.

The authors record one other case in which, again, the parents were both healthy but they were relatives; the patient herself was an intelligent child aged 8 years, again she revealed no other abnormality. On palpation nothing could be found in either orbital cavity.

E. E. CASS.

BOOK NOTICES

An Index of Treatment. Edited by ROBERT HUTCHISON. Eleventh Edition. Pp. 897, figures 147. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall, Limited. Price, 42s.

This work gives the practitioner a guide to therapeutics in a convenient form for reference. The articles are contributed by writers of accepted reputation. The subject matter contains what is known to be best, simplest and most effective in therapeutics and is not encumbered by a number of alternative procedures. The work has been brought up to date and in the 11th edition many articles have been entirely re-written and some added.

The sections on ophthalmic diseases have been admirably written by Sir William Lister and concern the management of the more common pathological disorders of the eyes. The author of this section has upheld the character of the book in its careful attention to facts that long experience has proved to be of value. Simple and sound conservative therapeutic principles are enunciated throughout this part of the work.

Many admirable illustrations are in the book, the printing, arrangement and reproduction of which are excellent.

Transactions of the American Ophthalmological Society. Vol. XXXIII. Philadelphia. 1935.

The 71st annual meeting of the American Ophthalmological Society was held at Hot Springs, Va., on June 5, 1935. The transactions contain many scientific contributions of interest, abstracts of which we hope to publish in the abstract section of the *Brit. Jl. of Ophthal.* in due course.

This publication also contains obituary notices of those members who have died during the past year; and lists of the officers and the Council, the President of the Society and its members. The minutes are also recorded. The printing, illustrations and reproduction are in accordance with the customary high standard of this work.

"Nordist Lärobok I Oftalmiatrik." Fourth Edition. Pp. 553, Illustrations, 268. Albert Bonniers Förlag, Svenska, Bokförlaget Stockholm, 1936. Price, 25 kroner.

This is a large and exhaustive text-book for students of ophthalmology. The work has been carefully revised, partially re-written and somewhat abbreviated. The Editors are the late Professor Fritz Ask of Lund University and Professor F. Berg of Upsala University.

The contributors are as follows:—Ask, Berg, Boström, Dalén, Ehlers, Enroth, Frieberg, Grönholm, Hagen, Holm, Höeg, Nordenson, Ploman, Rönne, Vannas, representing all three Scandinavian countries.

The editors have exercised great care that there has been no unnecessary overlapping of subjects and reiteration of ideas.

There is a very complete index of subjects, also an index of the chief authors referred to.

The binding, paper and type are all first class. The illustrations, many of them in colour, are as good as in most other leading text-books, but the iris colouring is decidedly Nordic blue. Two illustrations may be specially mentioned: one presents coloured plates of five degrees of choroidal atrophy as seen by the ophthalmoscope arranged in a vertical series, with a corresponding series of microscopic sections; another shows the various types of glaucomatous fields, and is most informative for the student.

The last three chapters deal with Accidents, Blindness, and a History of Ophthalmology respectively.

OBITUARY

ADOLPH BRONNER

DR. BRONNER, whose death occurred at Bradford on February 7, was one of the best known provincial members of the ophthalmic circle in this country in the closing years of last century and the early years of the present. Of German extraction, he was the son of Edward Bronner, M.D., of Bradford, who took a prominent part in founding the Royal Eye and Ear Hospital of that city. Born in 1860 he was educated at Bradford and the University of London. He spent a good deal of time abroad at German Universities and qualified M.D. at Heidelberg in 1884. On his return he became a clinical assistant at Moorfields and took the M.R.C.S.(Eng.) in 1885, before settling in his native city.

Bronner succeeded his father as surgeon to the Bradford Eye and Ear Hospital in 1886. The combination of ophthalmology and laryngology in one person was not uncommon 50 years ago, and Bronner combined the work at Bradford, for he became Laryngologist at the Bradford Royal Infirmary.

Bronner was a most popular consultant in the north. He was at one time president of the Bradford Medico-Chirurgical Society; and he was also president of the Leeds Medico-Chirurgical Society.

On retiring from practice Bronner was elected Consulting Surgeon to the two hospitals he had served so well; while his memory is enshrined in the Bradford Eye and Ear Hospital by a tablet, placed beneath that in memory of his father.

Of his work for laryngology this is not the place to say more than that he was secretary of the section of laryngology at the International Medical Congress at Rome in 1894.

Bronner joined the Ophthalmological Society of the United Kingdom in 1886 and served on the Council for a period of three years, early in the present century. He contributed many papers of clinical importance to the Transactions and frequently joined in discussions. The subjects of these papers covered a wide range in ophthalmic medicine and surgery.

NOTES

Lectures at Moorfields Eye Hospital

A COURSE of eight lectures on modern ophthalmic instruments is to be given by Mr. E. F. Fincham, F.Inst.P., at Moorfields Eye Hospital on certain Wednesdays in April, May and June, 1936.

Details will be found in our advertisement columns this month.

* * * *

Death

As we go to press we learn with deep regret of the death on March 12, of Dr. W. H. Wilmer of Washington. We hope to publish a memoir in a later number.

FUTURE ARRANGEMENTS

1936

April 2-4.—Ophthalmological Society of the United Kingdom (Annual Congress), in London.

April 3.—Midland Ophthalmological Society, at Nottingham Eye Infirmary.

May 22.—Midland Ophthalmological Society, at Addenbroke's Hospital, Cambridge.

June 12.—Royal Society of Medicine, Section of Ophthalmology (Annual Meeting).

October 6.—Midland Ophthalmological Society, at Birmingham Eye Hospital.

December 1.—Midland Ophthalmological Society, at Birmingham Eye Hospital.

CONTEMPORARY OPHTHALMIC LITERATURE

American Journal of Ophthalmology. *February, 1936.*

CRISP. Headache.
WOODS. Sympathetic ophthalmia.
LEBENSOHN. Scientific and practical considerations involved in the near-vision test with presentation of a practical and informative near-vision chart.
HARRISON and JULIANELLE. Studies on the infectivity of trachoma.
CHARLES and POST. Lipodystrophia progressiva with ocular complications.
BARKAN. Ophthalmic errors.
FRY. The relation of accommodation to the suppression of vision in one eye.
ABRAHAM. The nonsurgical treatment of nonparalytic strabismus.
WOODRUFF. Management of complications in the operation for senile cataract.

Archives d'Ophtalmologie. *February, 1936.*

DEJEAN. Ocular malformations of ependymal origin. The ependymomata of the retina.
ROLLET. The bed of pre-corneal liquid.
HIPPERT. Two cases of intermittent exophthalmos. Varicocele of the orbit.

Annales d'Oculistique. *January, 1936.*

MAGITOT, TILLÉ and DUBOIS-POULSEN. Keratitis of nasal origin.
CHARLINC. Syndrome of the nasal nerve from bacillary toxaemia.
DE JAEGER. Dermoid cyst with cranial malformation.
VIALLEFONT. The relation of the hypophysis and the retina.
COLLIN. Intra-capsular cataract extraction.

Bollettino d'Oculistica. *December, 1935.*

MOTOLESE. An uncommon case of sympathetic ophthalmitis.
ANELLI. Some cases of Krompecher's basilioma.
COZZA. Histo-clinical contribution to the study of sarcoma of the choroid, its extrinsic and general effects.
FIORE. Research on the action of derivatives of urea on the permeability of the haemato-ocular barrier.
PETRAGNANI. Rare benign new formations of the ciliary body.

January, 1936.

BIETTI. Pathological deposition of fat in the globe with particular regard to that following serious intra-bulbar inflammatory processes.
BIOZZI. Observations and research on Fuchs' heterochromia.

Klinische Monatsblätter für Augenheilkunde. *February, 1936.*

THIEL. Glaucoma.
BRONS. A case of successful operation for parasagittal meningioma of the left parietal lobe.
HAGEDOORN. Hydradenitic adenoma of Moll's gland.
DENIG. Evacuation by prolapse procedure.
ENRÍQUEZ. Further new investigations of the fundus by means of the slit-lamp.
GEORGARIOU. Alternating convergent strabismus.
VON SZILY and MACHEMER. Further contribution to the electrolytic treatment of retinal detachment.
PREVEC. A case of angiomatosis retinae.
MIRANDA. The diagnosis of diplopia.
BIRÓ. The causes of blindness. A statistical statement of 1,000 cases.
JESE Keratitis nummularis.
FAZAKAS. Sporotrichosis of the lower lacrymal canaliculus.

Archivos de Oftalmologia de Buenos Aires. *January, 1936.*

ZAVALIA, BALADO and OLIVA. Posterior meningo encephalocele of the orbit treated surgically.